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ON DISEASES

OF THE

LUNGS AND PLEURÆ

INCLUDING

CONSUMPTION

BY

R. DOUGLAS POWELL, M.D., Lond.

Fellow of the Royal College of Physicians; Physician to the Middlesex Hospital and Joint-Lecturer on Practical Medicine at the Medical School; Physician to the Hospital for Consumption and Diseases of the Chest at Brompton; Late Assistant Physician and Lecturer on Materia Medica at the Charing Cross Hospital.

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To

LIONEL SMITH BEALE, M.B., F.R.C.P., F.R.S.,

PROFESSOR OF MEDICINE AT KING'S COLLEGE,
PHYSICIAN TO KING'S COLLEGE HOSPITAL,

THIS BOOK IS INSCRIBED WITH THE AFFECTION AND
GREAT RESPECT OF

The Author.

PREFACE.

The present volume is an amplified edition of the work on *Consumption and on certain Diseases of the Lungs and Pleuræ*, which was published in 1878. The entire work has been reconsidered, and for the most part rewritten; and new chapters have been added on the Physical Examination of the Chest; on Asthma; on the Etiology of Phthisis; on the Complications of Phthisis; on the Surgical Treatment of Pulmonary Cavities; on Hydatid of the Lungs, and on Mediastinal Tumors. The prophylaxis and treatment of Consumption has, contrary to the general plan of the book, and with the view of avoiding repetitions, been dealt with in chapters apart from those in which the varieties of the disease are described.

The author trusts that no apology is needed for the comparatively large portion of the work which has been devoted to treatment. Only those measures of treatment, however, have been advocated of the value of which the author has been practically convinced, and many remedial measures have on this account been omitted or only barely alluded to. In thus exercising his discretion, the author has felt the double responsibility of, on the one hand, avoiding a multiplicity of remedies that would only prove an embarrassment to the earnest practitioner, and, on the other, of omitting nothing of real value in treatment.

The author is conscious of some failing in both these directions, especially in the section dealing with the climatic treatment of Phthisis; all that he can plead is that he has written up to the limits of his present convictions and experience; his responsibility is lightened by the knowledge of the recent works written by those able climatologists to whom he has alluded in the text.

The very friendly reception which his former work met with by his professional brethren, encourages the author to hope that some of the short-comings of the present book will be indulgently attributed to the increasing cares and interruptions under which it has been written.

The sincere thanks of the author are due to many friends who have helped him by suggestions and information in the course of his work, and acknowledgments are also due to his friend, Mr. J. Bland Sutton, for careful revision of proofs and preparation of Index.

62 WIMPOLE STREET.

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Drawings made by Mr. Edgar Thurston, from specimens prepared by Dr. Percy Kidd.

(*This plate appears also in the volume of the "Medico-Chirurgical Transactions" for* 1885).

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Drawings by Mr. E. Thurston, from preparations by Mr. Taylor.

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Preparation and drawing by Dr. Percy Kidd.

Fig. 1

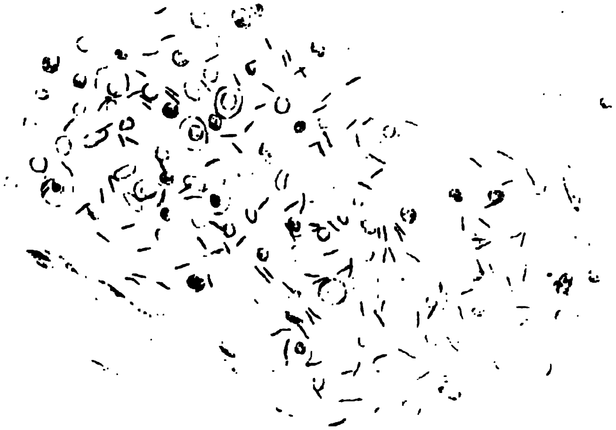

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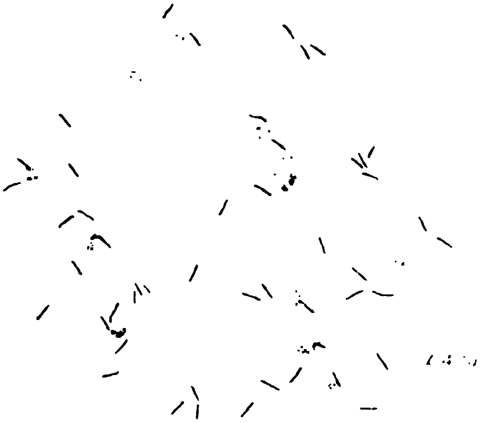
Fig. 1



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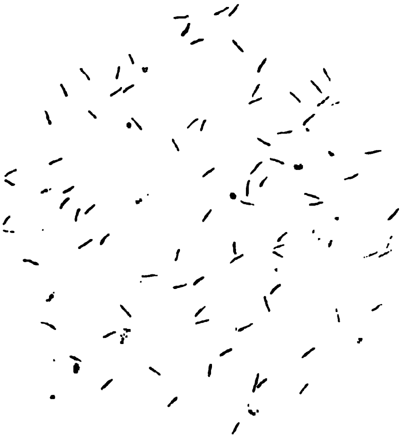

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DISEASES OF THE LUNGS AND PLEURÆ.

CHAPTER I.

ON SOME ESSENTIAL POINTS IN THE ANATOMY AND FUNCTIONS OF THE LUNGS.

BEFORE proceeding to discuss the pathology, detection and treatment of chest diseases, it will be well perhaps to recall to the recollection of the reader, certain essential features in the anatomy and configuration of the lungs, which are very practically concerned in the phenomena they present. Unfortunately, physiology is so widely separated from practical medicine in our schools, that the student has to relearn the one in its application to the other. Who for instance can rightly understand the phenomena of asthma, emphysema, pleuritic effusion, or pneumo-thorax, without a working knowledge of the statical and dynamical conditions of respiration in health. It was on the other hand complained by Addison, that morbid anatomy had not been sufficiently used to cast light upon the structure and function of parts in health, and it may perhaps be assumed, that the link between physiology and pathology is not so clearly observed nor so firmly maintained in our minds as it might well be. Let this reflection be my apology for devoting a chapter to certain physiological and anatomical considerations.

Anatomy.—Perhaps one could not better summarize the rough anatomy of the lung than by quoting the first four aphorisms of Addison respecting it, viz.: "1. That the aërial cellular tissue of the lungs is made up of well-defined, rounded, or oval lobules united to each other by interlobular cellular membrane, each lobule constituting a sort of distinct lung in miniature, having its own separate artery and vein; 2, that these lobules do not communicate directly with each other; 3, that they do not consist of the globular extremities of as many bronchial tubes, but are made up of a collection of cells in which, by a common opening, a minute filiform bronchial tube abruptly terminates; 4, that the pulmonary artery accompanies the bronchi, branch for branch, to the minutest divisions of the latter."[1]

---

[1] Observations on the Anatomy of the Lungs. Medico-Chirurgical Transactions, vol. xxiv., p. 146. Also New Sydenham Society's collection of Addison's Works, p. 2.

If we trace the bronchial tubes from the trachea into the recesses of the lungs, we find that in the course of subdivision they present certain important changes of structure. They divide in a peculiar way: 1, from the main bronchi down to the calibre of $\frac{1}{6}$ in. they divide symmetrically or dichotomously; 2, beyond $\frac{1}{6}$ in. the bronchi proceed in straight lines, gradually diminishing in size and giving off lateral branches alternately at an angle of 45°; 3, the lateral branches again divide dichotomously until they reach a diameter of about $\frac{1}{100}$ in., when they terminate in the *alveolar canals* or *passages*, the lobules referred to by Addison.

Down to a diameter of $\frac{1}{24}$ in. (1 mm.), the bronchial divisions are possessed of cartilages and muscular coat, are lined by a ciliated epithelium and are furnished with mucous glands. Their arterial supply is thus far derived from the bronchial artery, the capillaries of which terminate in the bronchial veins, forming a part therefore of the *systemic* circulation.

Below the diameter of $\frac{1}{24}$ in. (1 mm.), the bronchioles have neither cartilages, mucous glands, nor any continuous muscular coat; circular and longitudinal elastic fibres replace the muscular coat and the epithelium lining them is flattened. Their arterial supply is still derived from the bronchial artery, terminating however in the pulmonary venous capillaries and forming therefore a part of the *pulmonary* circulation.

The *alveolar canals* or *passages*, into which the ultimate bronchioles enter, are relatively large and immediately subdivide dichotomously two or three times, then terminate in somewhat bulbous ends—the infundibula. The first divisions of the alveolar canals are each somewhat larger than the ultimate bronchioles. The distance from the termination of the bronchiole to that of the infundibula, *i.e.*, to the extreme periphery of the lung, is from $\frac{1}{12}$ to $\frac{1}{6}$ of an inch.

The entire inner surface of the alveolar passages and their infundibular terminations is pitted by little secondary recesses, the alveoli, $\frac{1}{200}$ in. to $\frac{1}{150}$ in. diameter.

The alveolar passages and infundibula consist mainly of a framework of elastic fibrous tissue with a few unstriated muscular fibres; the alveoli or recesses within them are lined by a single layer of capillaries supported by a delicate membrane with elastic fibres, the elastic fibres forming bundles arranged round the openings of the alveolar recesses into the common infundibular passages; here the smooth muscular fibres are also found in greatest abundance. A delicate epithelium lines the alveoli.

Each lobule of the lung is thus made up of the branched alveolar passages and the alveoli opening into them, connected with one terminal bronchiole; it measures from about $\frac{1}{12}$ to $\frac{1}{6}$ in. in diameter.

With regard to the blood vessels of the lungs, it should be borne in mind that the pulmonary artery carrying dark blood, subdivides with the bronchial tubes and finally furnishes a capillary layer to each alveolus, each branch proceeding to its capillary distribution without communicating with any other branch. A junction between the pulmonary and systemic vessels is, however, effected at the pulmonary lobules by the discharge of the capillaries of the bronchial artery into those of the pulmonary vein as aforesaid. The veins have no valves, and for the most part take a separate course to the root of the lung in the interlobular or pulmonary fissures. They anastomose freely.

Respiratory function and mechanism.—In the recesses of the lungs within the alveoli and alveolar passages is effected that interchange of gases by means of which oxygen is on the one hand absorbed, and carbon dioxide (CO^2) exhaled, although no doubt the minutest bronchi allow of a certain oxygenation of blood through their fine membrane. The laws under which this interchange is carried on are those which regulate the diffusion of gases and their passage through moist membranes, but some physico-chemical processes are also involved which are probably somewhat complex. This is, so to speak, the nucleus or basis of the respiratory function, but in order that this function may go on uninterruptedly, it is obvious that special provisions must be made for a regulated renewal of the blood operated upon, and a thorough and sustained ventilation of the minute air chambers in which the process takes place. The rhythmic contractions of the right ventricle of the heart ensure a continuous current of blood through the alveolar capillaries, and the rhythmic respiratory movements secure, under conditions to be immediately stated, a thorough ventilation of the lungs.

Let us now glance at the mechanism by which these respiratory movements are effected. It need scarcely be observed that in the fœtus at the full term the lungs are in a state of complete and airless collapse, that the diaphragm is in a position of extreme convexity upwards, and the thorax in the position of utmost contraction, the respiratory function being effected by the interchange of gases between the fœtal capillary tufts of the placenta and the maternal blood in which they are steeped.

In the new-born child, however, on the placental circulation being interrupted, carbon dioxide accumulates in the blood and stimulates the respiratory nervous centres, thus exciting acts of inspiration. This central nervous stimulation would probably not alone be sufficient, but it is supplemented on the first exposure of the child to the external air, by a general excitation of the cutaneous nerves causing strong inspiratory movements. In the Gulstonian Lectures for 1872, Dr. Hensley conjectured that the first expansion or unfolding of the air cells of the lungs in the infant, was caused by the penetration of the blood through their capillaries: and this view was supported by some experiments made in the same year by Liebermann, who roughly imitated the structure of an air-sac by taking two ox-bladders, placing one within the other, and between them a layer of india-rubber tubing. On injecting oil through the tubing, the bag, previously collapsed, expanded, drawing in air with a perceptible sound.[1] Probably all these forces come into operation nearly simultaneously, and we thus get successive inspiratory expansions of the chest, in obedience to which the air penetrates the lungs, and is never again fully expelled.

It is not at all curious that the lungs, thus once inflated, should remain more or less permanently expanded, retaining in their interior a certain amount of air. They, like any ordinary elastic air bags, would do this, whether in or out of the body. But in the body, whether during life (within the limits of ordinary respiration), or after death the lungs are expanded *beyond* the point to which they would, from their mere elasticity, revert. This is a fact admitted by modern physiologists, but not, so far as I am aware, explained by them, nor is its importance in clinical medicine sufficiently recognized. I am, therefore, the more desirous to draw attention to it here.

We have no difficulty in understanding how, after the separation of the placenta, the gradual accumulation of carbon dioxide should excite the medullary centre to initiate respiratory movements, and how further excitation should be conveyed to this centre from the peripheral nerves, on the sudden contact of cool air with the surface of the body. Moreover, we may well believe that the physical effect of a diversion of the blood through the lungs would be to unfold the air vesicles, and thus enable them to become permeated with air. In order to account, however, for the maintenance of the lung in the semi-expanded state we must further assume that when the muscles of respiration, which are for the most part inspiratory muscles, have once been excited to action, they remain permanently shortened by virtue of their vital contractility or *tonus*. And in the course of tissue growth, the ribs and cartilages become moulded to that wider arc which they have thus been brought to assume.

The contractile power of the lungs derived from their residual tension, has been measured by different observers with somewhat different results. Dr. Carson, in 1820,[2] demonstrated the existence of this reserve tension, and estimated it in different animals as equivalent to from six to twenty, or more, inches of water, according to the size of the animal. He fully perceived the importance of this elastic force, both in the respiratory mechanism and as an aid to the circulation. Dr. Carson indeed regarded the respiratory movements as due to an antagonism between the elastic resilience of the lungs, and the muscular action of the diaphragm. He maintained that the traction of the lungs upon the diaphragm gave rise to a sense of uneasiness, to overcome which the muscle contracted, but that the enduring lung-elasticity on the cessation of the muscular effort again drew up the diaphragm. This view, although erroneous, was based upon an accurate observation of phenomena which were for a long time afterwards ignored.

---

[1] Wien. Med. Zeit. No. 5, 1872. I am indebted to Dr. Lauder Brunton for the reference to this experiment. Hensley's lecture is briefly alluded to in the Brit. Med. Journal, June 22, 1872.

[2] Philosophical Transactions, 1820, pt. i., p. 29.

Donders, in 1853,[1] made some experiments on the human subject. By connecting a manometer with the trachea, and then carefully opening the thorax, he was able to ascertain the residual elastic tension of the lungs, and concluded that in the healthy person it was equal to 80 millimeters (about three inches) of water. He further added 20 mm. for the tonicity of contractile elements in the lung.

Dr. Salter, in 1865,[2] found the residual tension in the dog equal to four inches of water.

Dr. M. Perls. in 1869,[3] gives the result of one hundred experiments, conducted in the same manner as those of Donders, upon persons who had died of different diseases: out of these, in 25 cases death had been caused by diseases remote from the lungs, although in most instances the lungs were in some degree affected. The highest degree of residual tension registered by Dr. Perls' manometer from these 25 cases was 60 mm. of water, the lowest 5 mm.: mean 35.3 mm.

It is very possible that the experiments of both Donders and Perls give results somewhat short of those of perfect health. It is, at all events, singular that the highest elastic pressure obtained by Perls from one of the 25 cases in which the lungs were presumably healthy, does not equal that (63 mm.) obtained from cases of bronchitis and phthisis respectively.

The amount of muscular tissue in the human lung is at most but very scanty. Donders added 20 mm. to his estimate of residual lung contractility on account of muscular tonicity. Stricker admits that here and there in the free margins of the alveolar passages nearest the bronchial terminations from which they are prolonged, delicate bands of smooth muscular fibre are to be found "which often consist of merely an isolated fibre imbedded in a delicate connective tissue. The membraneous walls of the alveoli themselves are entirely destitute of muscular tissue, nor have I been able to discover any muscular fibres in the more compact borders of the individual alveolar septa."[4] It seems that this tissue is more abundant in the lungs of animals. Müller found the lung of a dog contract on the application of iced water.[5]

Although in the human subject we may conclude that so far as the lung proper is concerned, muscular action takes no direct part in the respiratory mechanism, it is yet impossible not to see that the muscle-containing bronchial tubes which ramify throughout the lungs with whose texture they are in intimate union, must indirectly add to their contractility. We are more concerned now, however, with the purely elastic elements of the breathing apparatus.

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[1] Zeitschrift für Rationelle Medicin, N.F., bd. iii. (1853), p. 290.

[2] Lectures at the College of Physicians, Lancet, 1865, vol. ii., p. 142.

[3] Deutches Archiv für Klinische Medicin, bd. vi. (1869), p. 1.

[4] Human and Comparative Histology, New Syd. Soc. Edit., vol. ii., p. 60.

[5] Ludwig's Arbeiten, 1869, p. 64. Dr. Brunton informs me that this contractility was found to continue for two days if the lungs were supplied with blood by artificial circulation.

It is curious that whilst the residual tension of the lungs had been thus so carefully tested and measured, the thoracic tension necessarily equal and opposite to it should have almost escaped observation, or rather should have been deliberately misplaced as an expiratory force.

It was maintained by Hutchinson nearly forty years ago, in an able and elaborate paper read before the Royal Medical and Chirurgical Society of London,[1] that *inspiration is a wholly muscular act; the muscles in expanding the thorax having to contend against,* (1) *the elastic resistance of the lungs;* (2) *the inertia and elastic resistance of the chest walls. Expiration on the other hand being effected by the elastic recoil of the lungs and chest walls.* This view of the mechanism of healthy breathing has been accepted without question by the authors of most of our physiological text-books. Hutchinson estimated the resistance to each inspiration arising from the chest walls alone, at more than 100 lbs., and in deep inspiration at from 400 to 1000 lbs.

There are certain facts, however, many of which have been long known, which clearly show that *in quiet inspiration there is no inertia or elastic resistance of the chest walls to be overcome, but that on the contrary the thoracic elasticity is a reserve force of appreciable power constantly tending to enlarge the thorax, and therefore acting in favor of inspiration.*

That this must be so, would follow as a matter of reasoning from what has been observed above respecting the state of tension in which the lungs are maintained, so long as they remain healthy, throughout life; but more fully to demonstrate my proposition, I will briefly refer to some observations and experiments of other authors as well as my own.

The late Dr. Salter[2] was, I believe, the first to point out that at the commencement of breathing, the elasticity of the chest walls should be placed as an inspiratory force. By a simple experiment he showed that in the dead subject, the thorax, when relieved from the traction of the lungs by making an opening into the pleura, expanded to the extent of $\frac{1}{100}$ inch. Paul Bert[3] (1870) in some ingenious experiments, recorded simultaneously the elastic pressure of the lungs, and the expansion of the chest wall, at the moment of opening the pleura of a recently killed dog. Traube[4] in 1871, found that in the living dog the chest walls on being relieved from the traction of the lungs, expanded in the manner indicated by Salter; but Traube does not seem to have been aware either of Salter's or of Bert's experiments, and he erroneously asserts that the expansion which he attributes to muscular action, is not obtained in the dead animal. The table on page 8 contains the details of ten *post-mortem* examinations made by myself in 1876,[1] to estimate the extent to which the chest wall would expand of its own resilience, when relieved from the traction of the lungs. The difficulty of finding the thoracic organs perfectly healthy in the dead subject is well-known, and out of the ten cases operated upon in only four instances were they approximately so. The experiments were performed after the manner of Salter by taking a fine tube, A, expanded below and having stretched across its expanded extremity, B, a piece of caoutchouc provided with a projecting button, C. The instrument having been filled with colored fluid, was so adjusted that the button was accurately applied to the third cartilage, the integuments having been previously reflected from the front of the chest. The level of the fluid having been noted on the scale D attached to the stem, the thorax was cautiously opened and an immediate rise of the colored fluid was observed. A simple calculation enabled one to ascertain the actual

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[1] Vide Transactions of the Society, vol. xxix., 1846.

[2] Loc. cit.

[3] Leçons sur la Respiration, 1870, p. 359.

[4] Gesammelte Beiträge, bd. i.; Experimentelle Untersuchungen, 1871, p. 141.

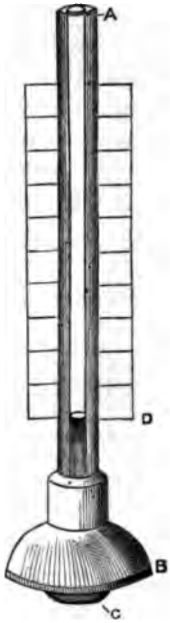

FIG. 1.

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[1] "On some effects of Lung Elasticity in Health and Disease." Medico-Chirurgical Transactions, vol. lix.

TABLE GIVING OBSERVATIONS ON THORACIC RESILIENCE IN TEN CASES POST-MORTEM.

All observations were made with the co-operation of Dr. Coupland at the Middlesex Hospital.

No.	Sex.	Age.	P.M.	Case.	Point of application of bulb.	Pleura opened.	Rise of fluid in stem. Centim.	Rise of fluid in stem. Total.	Equivalent expansion of Chest. Millim.	Remarks.
1	M.	33	24 hrs.	Amputation.	3rd L. cart.	*a*—2nd L. space *b*—2nd R. space	3.15 0	3.15	2.143	Right lung adherent, emphysematous; left free, slightly emphysematous; abdomen flaccid.
2	F.	38	21 hrs.	Tumor of jaw.	3rd mid. st.	*a*—3rd space *b*—3rd space	....	3.5	2.39	Slight further rise on making two fresh openings at 5th space; slight bronchitis.
3	M.	58	27 hrs.	Epithelioma of penis.	4th mid. st.	*a*—3rd R. space *b*—3rd R. space *c*—3rd L. space	0 0 2.4	2.4	1.63	Right lung adherent, side more prominent; some emphysema of both lungs, mostly of the right.
4	F.	44	20 hrs.	Carcinoma of uterus	5th mid. st.	*a*—2nd R. space *b*—2nd L. space	2.3 2.3	4.6	3.19	Lungs quite healthy, no adhesions; collapsed to about ⅕th of bulk; rigor mortis, no decomposition; abdomen flaccid.
5	M.	27	23 hrs.	Delirium tremens	4th mid. st.	*a*—3rd R. space *b*—2nd L. space	0 —0.3	—0.3	0	Œdema of both lungs, recent pleural adhesions both sides.
6	F.	36	....	Comp. fract., bruised strnm.	5th mid. st.	*a*—2nd R. space *b*—2nd L. space	0 0	0	0	Bronchitis; adhesions both sides.
7	M.	60	....	Fractured ribs, left side	4th mid. st.	*a*—2nd R. space *b*—2nd L. space	0 0	0	0	Cartilages ossified; right lung emphysematous; three pints of serum in left pleura.
8	M.	64	....	Strangulated hernia	4th mid. st.	*a*—2nd R. space *b*—2nd L. space	0.3 0	0.3	0	Lungs emphysematous and œdematous.
9	M.	41	12 hrs.	Fractured skull.	4th mid. st.	*a*—2nd R. space *b*—2nd L. space	0 0	0	0	Lungs highly œdematous; dynanometer connected with trachea registered no pressure.
10	F.	56	40 hrs.	Carcinoma of uterus	5th mid. st.	*a*—3rd L. space *b*—3rd R. space	0 —0.4	—0.4	0	Rigor mortis passing off; abdomen distended, chest-walls rigid, lungs emphysematous, but collapsed half an inch.

GENERAL REMARKS ON TABLE.—The first four cases give positive results, viz., an actual expansion of chest on opening the pleural cavities=from 1.6 mm. to 3.19 mm. Case 4, giving the greatest expansion, was the only case in which the lungs were quite healthy. The last 6 cases give practically negative results, viz. no expansion or contraction in 3 cases, a small fractional expansion in 1 case, and a small fractional contraction in 2 cases. In all these latter 6 cases the lungs were notably affected by disease.

expansion registered. The results of the experiments will be seen in the table. In the four comparatively healthy cases the expansions measured were 1.63 mm., 2.143 mm., 2.39 mm., and 3.19 mm. respectively.[1] These figures, small though they are, indicate pretty well the limits of expansion of the ribs during calm breathing. Thus Hutchinson calculated the costal movement in health at ⅒ to ¼ line=1 to 2 mm.; Sanderson gives 1.6 mm. My own measurements would give rather a higher figure, viz., 2 to 3 mm. But from some careful tape measurements I have found the total circumferential expansion in health (in quiet breathing) not to exceed ⅛ to ¼ in. (4 to 8 mm.), in some exceptional cases ⅓ to ½ in. Age, sex, and many other conditions influence the mobility of the thorax. It would appear then—and this is the point to which I wish especially to direct attention—*that in health throughout ordinary inspiration, the limit of thoracic recoil, which we may call the reserve capacity of the thorax, is barely reached; and therefore that the sole resistance to be overcome by the inspiratory muscles is that of the lungs.*

In order more clearly to show the main physical conditions present in the chest and how they are modified, (*a*) during normal respiratory movements, and (*b*) in certain diseases, the subjoined schema or diagram-model of the chest was designed.

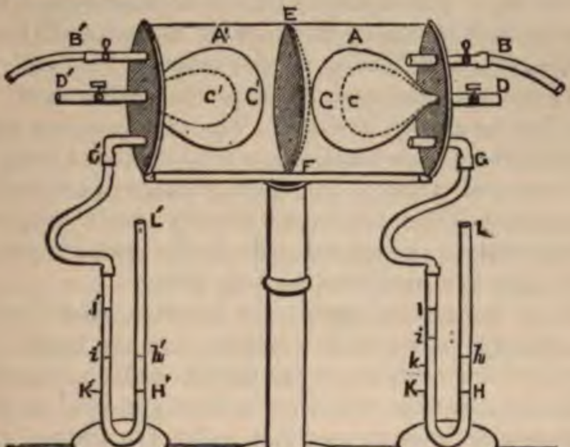

Fig. 2.

The schema consists of a cylinder of glass, closed at each end by a metal plate screwed on and perforated for the admission of certain tubes.

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[1] For fuller details respecting these and other experiments see the original paper, "On some effects of Lung Elasticity in Health and Disease," by the author in the Medico-Chirurgical Transactions, vol. lix.

A central partition, EF, made of sheet india-rubber, divides the cylinder into two air-tight compartments, each of which, as will be presently seen, represents one half of the thorax.

Tube D represents the trachea, and is connected with an elastic bag, (*c*) representing the (left) lung. Tube G communicates with the space between the lung and the wall of the chamber, which space, therefore, corresponds to the pleural cavity. This tube is connected with a mercurial manometer, the free end of which, L, is open to the normal atmospheric pressure. Tube B also communicates with the same (pleural) space, and is provided with a stop-cock and a mouthpiece.

Exactly the same parts are repeated on the opposite side of the partition, EF, which therefore represents the mediastinum.

The apparatus must be ascertained to be thoroughly air-tight. Then by partially exhausting the air from chamber A through tube B (trachea tubes D and D′ remaining open) we cause the bag *c* to expand to C, the mercury in the manometer, HKI, to rise towards the chamber, and the mediastinum, EF, to become convex, as indicated by the dotted line. By closing the stop-cock B we maintain all the parts in this position.

If, next, we repeat the same process on the opposite side—partially exhausting chamber A′ through B′ until the mercury I′ is at the same level as I—the bag *c*′ will expand to C′, the mediastinum, EF, will again become vertical, and by closing stop-cock B′ the parts will be maintained in this position of equilibrium on the two sides.

In the schema thus arranged, we have the conditions of the healthy chest rudely but accurately imitated. The two chambers represent the two sides of the chest, each containing a semi-expanded lung, C, C′, surrounded by a pleural cavity, A, A′, (here greatly exaggerated,[1] the cavity being rather potential than real in the healthy chest), each cavity being separated from that on the opposite side by the mediastinum EF, which is common to both and equipoised between them.

The walls of the natural thorax are, however, elastic or resilient in every part, although much more stiffly so than the lungs. We cannot exactly represent this resilience of the thoracic walls in our schema. The only parts of our apparatus which are at liberty to yield to the excess of external atmospheric pressure over that within the pleuræ, are the small surfaces of mercury at H,H′. Hence the elevation of the mercury HI, H′I′, towards the chamber on each side, multiplied by the area of apertures G,G′, and divided by that of the whole surface of the chamber, would represent in millimeters the amount of recession of each portion of the thorax, provided each portion were equally resilient.

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[1] This is unavoidable, since the walls of the schema are rigid, and if the bags fitted accurately, their further expansion in inspiration could not be represented. No fallacy is hereby introduced, however.

Although in the natural chest, the diaphragm yields far more than any other portion of the chest wall to the traction of the lungs; this is chiefly because its resistance is weaker, so that a recession of an inch on the part of the diaphragm is only equivalent to the recession of one or two millimeters on the part of the ribs or cartilages. For it must be further remembered, that the diaphragm is not elastic, and that the limits of its *tension* are therefore very narrow; and indeed, the elastic recoil of the diaphragm depends mainly upon the spring of the cartilages to which it is attached. Our mercurial columns therefore, after all, very conveniently and fairly represent the whole thoracic resilience in a lump sum.

Having thus with the aid of our schema rehearsed the statical conditions of the chest, the dynamics of respiration may be easily demonstrated.

In order to imitate an inspiration, aspiration must be made simultaneously through the tubes L and L′, thus representing the contraction of the diaphragm and inspiratory muscles on the two sides. This can be conveniently done by means of a syringe attached to a branched caoutchouc tube affixed to the extremities L L′ of the manometers.

As the mercurial columns rise up the limbs L L′ of the manometers, representing the expansion of the thorax, the lung C C′ enlarge by the entry of air through trachea tubes D and D′.

It will be observed that, during the first part at least of inspiration, the weight of the two columns of mercury H I, H′ I′ tells in favor of the inspiration. This weight of mercury corresponds as before said to the outward resilience of the thoracic walls, and counter-balances the elastic traction of the lungs. This is a fact, well shown in the schema, and which has been already stated in the proposition, that *in health the resilience of the chest wall is in favor of inspiration.* Moreover, from the observations on the dead subject to which I have already referred, it would appear that *this elastic aid to inspiration obtains throughout the act in calm breathing.* Respiration is thus rendered smoother and less laborious, elasticity entering as an important item into the inspiratory, as it has been long known to do into the expiratory, act.

The conditions present in the chest as shown by the schema must be further examined in their effects upon the heart and circulation.

The disposition to the formation of a vacuum in the intra-pleural space A, equal to the weight of the column of mercury H I, causes an aspiration towards that cavity which was at first shown by the convexity of the mediastinum E F (dotted line): and there being a similar and equal aspiration towards the pleural cavity on the opposite side of the mediastinum, it follows that there is a constant determination of blood towards the cavities and walls of the heart—a hollow organ situated within the mediastinum, and communicating by a system of tubes with parts outside the thorax, (see fig. 2). This central attraction is forcibly overcome by the muscular contraction of the heart, but resumes its sway at the termination of systole, aiding the return of blood to the flaccid heart cavities, and encouraging the flow through the coronary vessels. This aspiration towards the heart is, be it remembered, in health a constant force, increased during inspiration, held in momentary abeyance during the more forcible muscular contraction of the heart, but not wholly extinguished even at the end of ordinary expiration.

In the drawing on page 13, which depicts a somewhat more elaborate diagram-model of the chest (but essentially corresponding with that illustrated by fig. 2), the mediastinum is represented by a double layer of caoutchouc enclosing a space, connected with which the manometer, D, shows at a glance the negative pressure to which the heart is constantly subjected through the traction of the lungs from either side.

In discussing, however, what has been called the statics of respiration, we must not leave the diaphragm out of account. It is obvious that this muscular membrane must be drawn upwards and held in a state of tension by the lungs during the respiratory pause. Mr. Le Gros Clark, in a paper read before the Royal Society in May, 1871,[1] demonstrated what he termed the passive tension of the diaphragm, and the observations of Traube,[2] and more recently those of G. M. Garland of New York,[3] show how this elastic traction upon the diaphragm importantly modifies the physical signs of pleuritic effusions.

The capacity and willingness (if I may so say) of the thorax to enlarge within certain limits, without muscular effort from without or pressure from within, is a most valuable safeguard in some diseases, serving to protect the lungs from pressure during temporary conditions of inflammation or engorgement.

If we look at a case of pneumonia, we find that, whereas on the healthy side the play of the chest-wall is natural or increased, on the other side it is diminished. There is no retreat with expiration, and inspiration is checked by pain. On post-mortem inspection, we find the fine granulations of lymph evenly spread upon the pleural surfaces, showing no sign of pressure. The patient suffers little pain, except when he coughs or attempts to draw a deep breath. In fact, as the inflamed lung increases in bulk, the chest wall retreats to the position of inspiration; and this reserve capacity of the thorax is rarely exhausted in pneumonia, so that the swollen and tender lung as a rule escapes compression.

In temporary [illegible] of the lungs, the consequences would be much more serious were it not for the reserve capacity of the thorax, which protects the organs from compression, save in extreme

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[1] Proceedings of the Royal Society, vol. xx, p. 122.

[2] Gesammelte Beiträge [illegible] by Fraentzel in Ziemssen's Cyclopædia, English [illegible]

[3] Pneumono-Dynamics, by G. M. Garland, M.D., New York, 1878, p. 51.

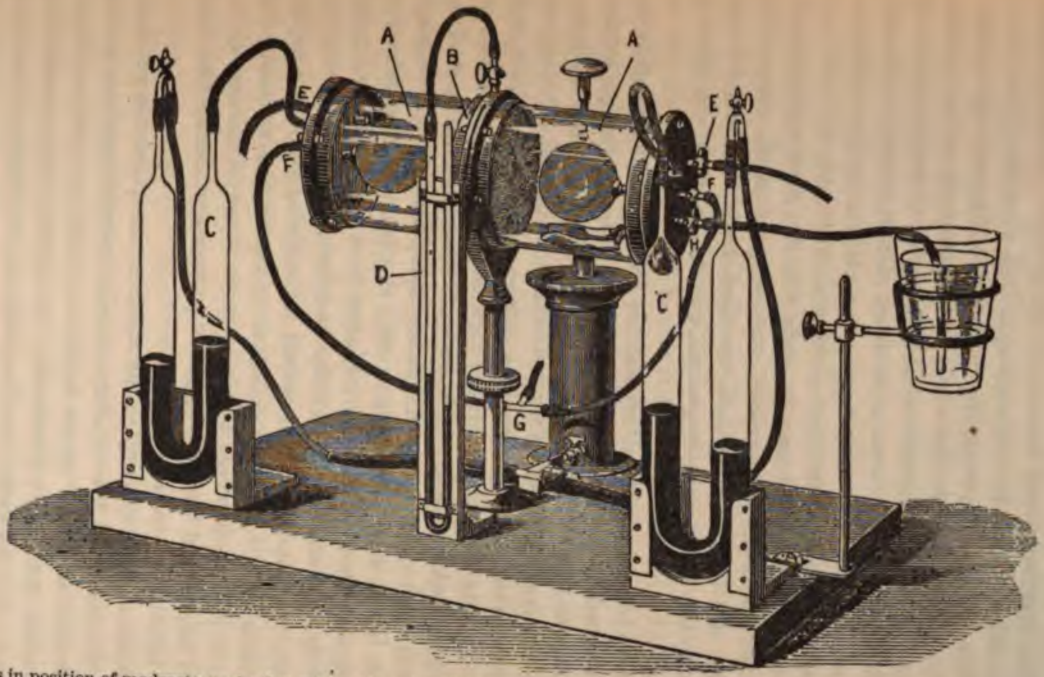

FIG. 3.—A A. Lungs in position of moderate expiration within pleural cavities.
B. Mediastinal space (pericardium).
C C. Manometers showing elastic traction of lungs upon chest walls.
D. Manometer showing negative pressure corresponding with traction of lungs within pericardium as CC show negative pressure within pleural sacs.
E E. Tubes with stop-cock (shut) communicating with pleuræ.
F F. Bronchial tubes uniting in G. Trachea.
H. Tube communicating with separate bag placed in pleural cavity A, for convenience in representing pleuritic effusions.

The instrument is represented as arranged by exhausting the air equally in the two chambers A and A to show the condition of respiratory statics in the position of respiratory pause. This is effected either by exhausting the air equally in the two chambers A and A until the bladders representing the lungs become partially inflated, or (what amounts to the same thing) by inflating the two lungs through the trachea, whilst stop-cocks E E are open, then closing these cocks and allowing the lungs to collapse to the point of equilibrium.

cases. Again, it is only in very extreme cases of emphysema that the eccentric thoracic resilience is entirely neutralized. I have directed my attention especially to this point in many cases, and I believe that the lungs are never enlarged so as to be compressed by the ribs.

With regard to *emphysema*, the loss of the elastic spring of the chest-wall is one of the first important features of the disease. In the normal chest, a deep breath is drawn by first making a deep expiration, and then a deep inspiration. In emphysema, the power of making a deep expiration is partially or entirely lost; the whole reserve capacity of the thorax is taken up, and it is in this direction that the trouble of emphysematous people lies. Regarding emphysema as in many cases essentially a disease of the lungs of a degenerative kind, we must observe in the constant traction of the thorax upon the lungs a determining cause of their enlargement. If, as is often the case in early life, the chest-walls be soft and feeble, they cannot properly expand, and we get a small, narrow, or distorted chest. On the other hand, if the elasticity of the lungs be relaxed by disease, the chest expands gradually to the limits of its reserve capacity.

Lymphatics.—I have preferred to pass on from the consideration of the general structure of the lungs to a discussion of the mechanism of the respiratory function, and even by way of illustration to refer to some morbid states of perverted respiratory mechanism, before completing our anatomical description of the lungs, by speaking of their lymphatic and nerve supply. I have done this with the view of bringing together these points of anatomy and function which are most concerned in the production of physical signs and symptoms. In the lymphatic and nervous apparatus of the lungs we have, so to speak, the drainage and function regulating systems, whose workings are hidden from us for the most part, to be brought to light mainly through the manifestations of disease. In truth, however, the important part played in pathology by the complex lymphatic system of the lung as disclosed to us by the researches of Sanderson[1] and Klein,[2] is becoming daily more clearly discerned as we become better acquainted with the origin of pulmonary diseases and their extension by infective processes.

If we commence our scrutiny in the walls of the pulmonary alveoli, we find that the spaces of the capillary vascular network contain, indeed constitute, lacunæ or lymph spaces which are lined or loosely occupied with protoplasmic connective-tissue cells: the lacunæ intercommunicate by fine processes and discharge into lymphatic vessels which, in the depth of the lung, accompany the minutest subcapillary blood-vessels (perivascular lymphatics), but which, on the surface of the lung, occupy the grooves between the lobules (sub-pleural lymphatics). The connective-tissue cells lining the lacunæ in the alveolar walls, send fine protoplasmic processes, which insinuate between the epithelial cells of the alveolar surface (pseudo-stomata), and thus maintain the lymphatic system in living contact with the air spaces. Dr. Hamilton[1] describes and figures some granular polygonal cells lying singly or in groups upon depressions of the alveolar surface, but denies their resemblance to pseudo-stomatous cells. "Although (remarks Dr. Hamilton, p. 106), there seems to be little evidence that these young granular embryonic cells are of a connective-tissue type, yet that they have a pseudo-stomatous action in transferring foreign bodies from the alveolar cavity to an underlying lymphatic, as claimed by Klein and others, can be verified."

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[1] Tenth and Eleventh Reports, Medical Officer of Privy Council.

[2] The Anatomy of the Lymphatic System, part ii.—The Lung.

The sub-pleural lymphatics inosculate by deep branches with the perivascular lymphatics, and further communicate directly with the pleura by means of fine canals which open upon its surface (stomata). Both these sets of lymphatics course respectively onwards to the bronchial glands. The bronchial tubes also give rise to lymphatics (peri-bronchial), which have their rootlets in fine canals and intercommunicating spaces in the mucosa, in direct communication with the mucous surface by protoplasmic processes of connective tissue cells projected between the columnar epithelial cells. These bronchial rootlets discharge into the larger lymphatic vessels of the adventitia, which, like the rest, course onward towards the bronchial glands at the root of the lung. The peri-vascular and peri-bronchial lymphatics, being adjacent, freely inosculate, especially at their finer divisions in the depth of the lung. Connected with the peribronchial lymphatics distinct acini or rudimentary glands have been discovered by Prof. Burdon Sanderson in the lung of the guinea pig, and it is reasonable to suppose that they are also represented in the human lung, although their existence has not as yet been demonstrated. The whole lung is thus pervaded in every crevice of its structure by lymphatic tissue, consisting of branched protoplasmic cells communicating with fine tubes and interstitial spaces occupied or lined with endothelial cells.

Nerves.—The nerve supply to the lungs is derived from the anterior and posterior pulmonary plexuses, which are formed by the interlacement of branches from the vagi, joined by others from the sympathetic, chiefly from the second, third, and fourth thoracic ganglia. The nerves from these plexuses accompany the bronchi, but their exact mode of termination is as yet undetermined. It seems, however, probable from the researches of Remak, Stirling[2] and others, that they terminate in the muscular fibres of the walls of the minute bronchi, and in their course present ganglia singly and in groups. One may with tolerable safety infer further that twigs are communicated to the ultimate lobules of the lungs.

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[1] Pathology of Bronchitis, etc., 1883, p. 104.

[2] Quain's Anatomy, 9th edit., vol. ii., p. 521.

CHAPTER II.

THE PHYSICAL EXAMINATION OF THE CHEST.

BEFORE proceeding to physical examination, preliminary inquiries have to be made into the health history of the patient, the circumstances which have led up to his present illness and the chief symptoms which give him distress. In the course of these inquiries the appearance and manner of the patient may be noticed; and the experienced physician, practically skilled in the physiognomy of disease, will thus gain information of great value, as a clue to the often confused story of the patient, and in suggesting to him further questions.

It would be folly to attempt to learn from a book the physiognomy of disease: clinical study, an habitually careful scrutiny of the features and postures of actual sufferers will alone impart it. The student will early recognize the aspect of turgid lividity and labored breathing of chronic bronchitis with dilated heart, from the pallor, anxiety, throbbing vessels, and dyspnœa with restlessness of advanced aortic regurgitant disease. The hectic look of phthisis, the grave drawn lineaments of asthma, the swollen features and cyanosed mucous membranes in heart disease, the puffy pallor of albuminuria, may in their more marked degrees be soon recognized even by the beginner. The finer traits and markings of disease, however, require more experience for their detection, and are of even more value in suggesting inquiries and often in leading one to suspect the presence of disease before sufficient signs can be found to justify a positive diagnosis. Incipient tuberculosis and obscure aneurysm may be named as two instances in which the features of illness sometimes suggest a more guarded diagnosis than the actually discoverable signs would warrant.

The relationship between physical signs and the diagnosis of pulmonary disease is again of a strictly practical kind, although of course depending upon acoustic principles, and he who would become a successful auscultator, *i.e.*, a good diagnostician, must study auscultation in association with morbid anatomy. Thus will the stethoscope reveal to him at the bedside, in most cases, an accurate picture of the lung, heart or pleura under examination, as though the organ were exposed to his view. This intimate association in the mind between physical signs and the lesions which give rise to them is only to be acquired by genuine clinical and *post-mortem* observation; no amount of reading or clinical work alone will suffice for its attainment.

It is the object of a text-book, however, to supply certain data for comparison and to lay down principles and methods upon which a satisfactory exploration of the chest is best founded.

Shape of the Chest.—It is impossible in a word to describe the shape of the chest, but it may be said in the adult to be conical from above downwards, flattened in front and grooved in the posterior median line, so that the antero-posterior diameter is about one-third less than the transverse. In the child these two diameters are more nearly equal. The shape of the upper portion of the chest is obscured by the pectoral muscles, extending from the upper arm to the clavicle and ribs, giving it a somewhat square outline. In the female the apparent form of the thorax is still further altered by the mammary development.

Measurements of the Chest.—Two instruments are of value for clinical use in the measurement of the chest, *viz.*, the *double tapes*[1] consisting of tapes connected by a central piece for adaptation to the spine, from which point each tape is graduated. The back piece being carefully held over the spinous processes and a mark being made exactly in the median line of the sternum, the two tapes are brought round the chest at the same level and with moderate firmness of application, to overlap one another at the front line. A comparative measurement of the two sides is thus at once read off, the sum of the two measurements giving the total circumference.

By holding the tapes lightly the expansion of the chest can be measured during calm breathing, and by making the patient take a deep breath followed by a full expiration the total expansibility is ascertained, also, what is equally of value, the contractility of the chest beyond the point of ordinary expiration. Finally, a comparison in all these respects can be made between the two sides.

By means of the *cyrtometer*[2] a tracing of the circumferential outline of the chest is obtained. This instrument consists of two pieces of thin lead piping connected by a hinge of rubber tubing. The hinge is carefully applied over the spinous process at the level required, and the piping moulded round the chest until the two ends cross one another in the median line in front; a mark is then made and the instrument allowed to fall away from the chest, held by the flexible joint; it is subsequently adjusted in position on a sheet of paper, and "sternum" and "spine" being marked, the pencil is carried round the inner circumference, and an exact outline of the shape of the chest is thus obtained, any comparative or general alteration in shape being readily observed.[3]

The circumference of the chest varies much in different individuals and within the range of health. Thus, Walshe[4] has noted in adult males of medium height, measurements ranging between 27 inches and 44 inches in the circumference, taken opposite the sixth rib. He would regard 33 inches as a fair average in the adult. Dr. Sieveking,[1] on the other hand, places the average measurement above the nipples as 38 inches. The minimum chest measurement for a recruit in this country is 34 inches, except he be under twenty years of age, and a growing lad, when an inch less is accepted.

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[1] First introduced into clinical use by Dr. Charles J. Hare.

[2] First used clinically in the form of jointed whalebone by M. Woillez. (Walshe's Diseases of Lungs, 4th edit., p. 33).

[3] The use of callipers for taking comparative antero-posterior measurements is attended with so many practical difficulties as to be of little value for clinical purposes.

[4] Diseases of the Lungs, 4th edit., p. 30.

For clinical purposes these absolute measurements do not teach us much. Relative measurements of the two sides are of value in certain diseases attended with enlargement or diminution of the chest on one side. Cyrtometer tracings are of most value since they give exact information as to shape. The mobility of the chest is of more importance than its mere size, as it affords a better indication of vital capacity. In the healthy adult the difference between extreme inspiration and extreme expiration should not be less than 2½ inches, as measured by the tapes at or about the level of the nipples. It may amount to as much as five inches. (Walshe.) The difference should be nearly equally divided between the two sides, a slight excess in favor of the right side being of no account.

In calm breathing, however, the actual movement of the chest is very small, averaging in the healthy male (according to Walshe) ¼ inch: the movement upwards of any one spot of the chest surface not exceeding two to four millimetres.[2]

Pneumatometry.—The power ordinarily employed by the inspiratory and expiratory forces during calm breathing, and that which they are capable of exercising during extremest effort, have been carefully estimated by various authors, and most recently by Waldenburg.[3]

The instrument employed by Waldenburg for this purpose consists of a manometer provided with a naso-oral mask so padded as to fit with accuracy, each limb of the manometer measuring about twelve inches (270 mm.), and being half filled with mercury.

For calm breathing the mercurial surface would indicate a difference of from one to two millimeters. With forced inspiratory effort the mercury could be maintained at a minus pressure of 2½ inches (60 mm.), nearly double this pressure being momentarily attainable.

The expiratory force exceeded this inspiratory by twenty to thirty mm., *i.e.*, with forced expiratory effort 3½ inches (90 mm.), of positive mercurial pressure might be maintained and, momentarily, as much again. Marked variations from the healthy standard are met with in disease, and in two directions:—

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[1] Medical Adviser in Life Assurance, p. 140.

[2] Medico-Chirurgical Transactions, vol. lix., p. 169. The whole subject of mensuration will be found fully discussed in Walshe's treatise. For detailed researches respecting the movements of individual ribs consult Dr. Ransome's admirable work on Stethometry.

[3] The subject of pneumatometry and spirometry is most fully and ably discussed by Waldenburg in his work, Die pneumatische Behandlung der Respirations- und Circulations-krankheiten. Berlin, 1880.

(1.) *The inspiratory power is diminished whilst the expiratory (except in extreme cases) remains normal.* This type is observed in phthisis, even in the earliest stages of that disease. It is found also in laryngeal, tracheal and bronchial obstructions, and to a less extent in pneumonia or in pleuritic effusion.

(2.) *The expiratory pressure is lowered, the inspiratory remaining normal or being even increased, or sub-normal, but in all cases remaining relatively higher than the expiratory.* This type obtains in emphysema, bronchitis and asthma, also in diseases of the abdominal organs which impede the play of the expiratory muscles.

Spirometry.—In the healthy adult at rest the respirations number from sixteen to twenty per minute, they are somewhat slower during sleep than when awake, and are readily accelerated by movements, effort, or excitement of any kind.

There is an influx and efflux of about 30 cubic inches (500 cc.), of air (tidal) with each act of calm breathing, about 100 cubic inches (1,600 cc.), remaining in the lungs as reserve and residual air. By the deepest expiration following a full inspiration, a healthy man of average build can expel from 200 to 250 cubic inches (3000 to 4000 cc.), which represents the "vital capacity of the individual," leaving still behind in the lungs a certain amount of "residual" air which cannot be removed by any expiratory effort.

These facts were originally worked out by Hutchinson[1] by means of an instrument called the spirometer, which consisted essentially of a graduated gasometer nicely balanced and provided with a mouth piece, through which the patient could breathe into the meter previously set at zero. By a series of very elaborate investigations Hutchinson arrived at the following important conclusions which have not been altered or materially added to since his original paper.

Hutchinson found that the vital capacity varied with the height in a very definite manner as will be seen by the subjoined table.

Height.	Vital Capacity. Hutchinson.		Weight.	Circumference of Chest. Allen.[2]
	From observation.	From calculation.		
ft. in. ft. in.	cub. in.	cub. in.		
5 0 to 5 1	174	174	120 lbs.	34.06 in.
5 1 " 5 2	177	182	126 "	35.13 "
5 2 " 5 3	189	190	133 "	35.70 "
5 3 " 5 4	193	198	136 "	36.26 "
5 4 " 5 5	201	206	142 "	36.83 "
5 5 " 5 6	214	214	145 "	37.50 "
5 6 " 5 7	229	222	148 "	38.16 "
5 7 " 5 8	228	230	155 "	38.53 "
5 8 " 5 9	237	238	162 "	39.10 "
5 9 " 5 10	246	246	169 "	39.66 "
5 10 " 5 11	247	254	174 "	40.23 "
5 11 " 6 0	259	262	178 "	40.80 "

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[1] Medico-Chirurgical Transactions, vol. xxix., 1846.

[2] Med. Exam. for Life Ins. N.Y., 1872. Quoted by Dr. Sieveking, loc. cit., p. 141.

In the preceding table the two columns of vital capacity, one taken from a number of observations, the other from calculation, are so nearly identical that we may take it that every inch in stature above five feet should add eight inches to the vital capacity. The last column has been added to show the corresponding circumferential measurements of the chest.

The body *weight* also influences the vital capacity; for example, at the height of five feet six inches the vital capacity decreases one cubic inch per lb., from 161 lbs. (an excessive weight as will be seen for the height) to 196 lbs.

After thirty and up to sixty years of age there is a decrease of nearly 1½ c. in., per year of age. In disease the vital capacity decreases from ten to seventy per cent. This diminution is dependent upon, but not directly proportional to, the extent of breathing surface encroached upon, since the lung or portion of lung remaining healthy may take a compensatory action. (Waldenburg).

Topography of the Chest.—For convenience in clinical examination and description the chest is mapped out into certain regions; these are sufficiently indicated by the terms employed, viz:—

Anteriorly, the supra-clavicular, clavicular, infra-clavicular, mammary and infra-mammary regions on the right and left sides respectively.

In the median line, the supra-sternal, upper sternal and lower sternal regions.

Laterally, the axillary and infra-axillary regions.

Posteriorly the upper scapular (supra-spinous), the lower scapular (infra-spinous), the inter-scapular and the basic regions on each side.

For the purpose of more accurately noting for future reference, the locality of any particular physical signs, there is no better or simpler plan than that of employing imaginary lines and levels drawn upon the chest surface. Thus the chest can be mapped out in latitude and longitude by parallel *vertical* lines drawn from summit to base through the *mid-sternal, para-sternal, sterno-nipple, nipple, anterior axillary, mid-axillary, posterior axillary, mid-scapular, interscapular* and *vertebral lines,* intersected by parallel lines drawn *horizontally* at the levels of the several rib cartilages in front and the several spinous processes behind, with the addition of *nipple level, ensiform level,* etc.

A careful observation of the adjoining diagrams will impress upon the memory, the main features in the topography of the chest organs and of those abdominal viscera in immediate relation with the chest.

Marginal references are made at certain levels to facts of topography, which it is of some importance to bear in mind in clinical work. It will be observed that the upper or anterior lobes of the lungs occupy most of the front aspect of the chest, whereas posteriorly the lower or posterior lobes correspond with nearly the whole surface. The division between the two lobes takes place on either side close to the spine at the level of the spinous process of the third dorsal vertebra, and the line of separation slants downwards and outwards, across the junction of the middle and lower thirds of the scapula and the sixth rib in the axillary line. On the left side it courses along this rib to terminate at its upper border in the nipple line; on the right side, having reached the sixth rib in the axilla, the line of separation continues downwards to the eighth rib, a second cleft passing almost transversely forward opposite the fourth space, to terminate in the median line at the junction of the sixth cartilage with the sternum; thus marking off the third lobe, which is interpolated in front between the upper or anterior, and the lower or posterior, lobe.

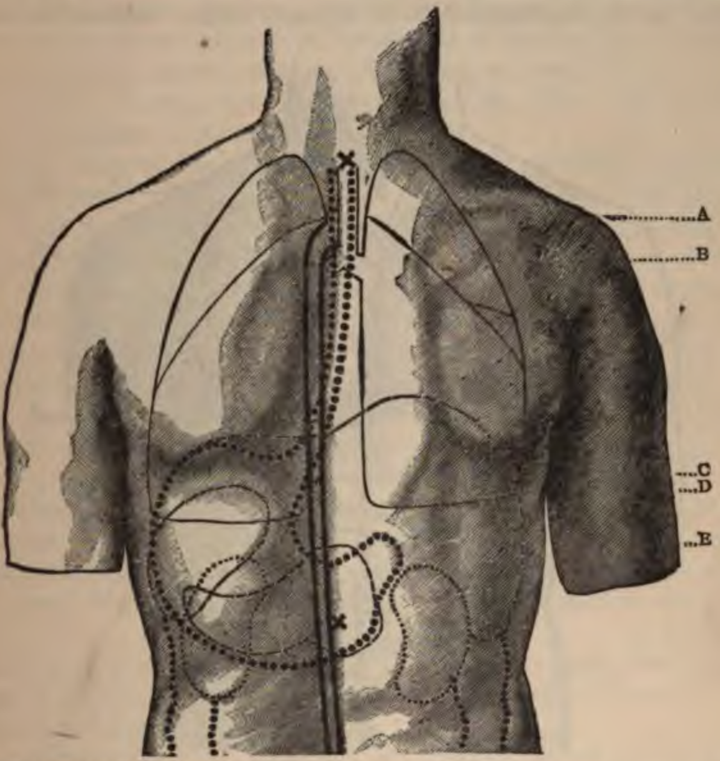

Fig. 4 (from Quain's Anatomy)—X 7th cervical spine = apex of lung.
A. Division between the lobes = tip of spine of 3rd vertebra, *i.e.*, 3 inches below summit of lung.
B. Division of bronchi = 4th dorsal spine.
C. Long axis of spleen = 10th rib.
E. Base of right lung = 10th dorsal spine. Base of left lung = 11th spine (base of this lung in diagram represented too high).
D. Upper end of left kidney = 11th dorsal spine, the right being ½ inch lower.
X. = 1st lumbar spine.

In physically exploring the chest, *inspection, palpation, percussion,* and *auscultation* are successively employed.

Inspection.—The chest should always, if possible, be uncovered, so that a general view of its conformation can be obtained, whether it be the broad, well-formed chest of robust health; or the small, narrow, long chest—with antero-posterior and lateral diameters diminished, costal angle narrowed, and ribs unduly oblique and approximated—adapted to small lungs; or whether the thorax be unduly expanded with widened intercostal spaces, straightened ribs, increased costal angle, and deepened antero-posterior diameter—making up the round-shouldered barrel-shaped chest adapted to the accommodation of enlarged lungs. Again, the thorax may be distorted by various kinds of spinal curvature, by rickets in early life, or by continued pressure in any particular direction. Finally, there may be local flattenings or bulgings of the chest walls, due to alterations in the subjacent viscera, and giving rise to a want of symmetry on the two sides.

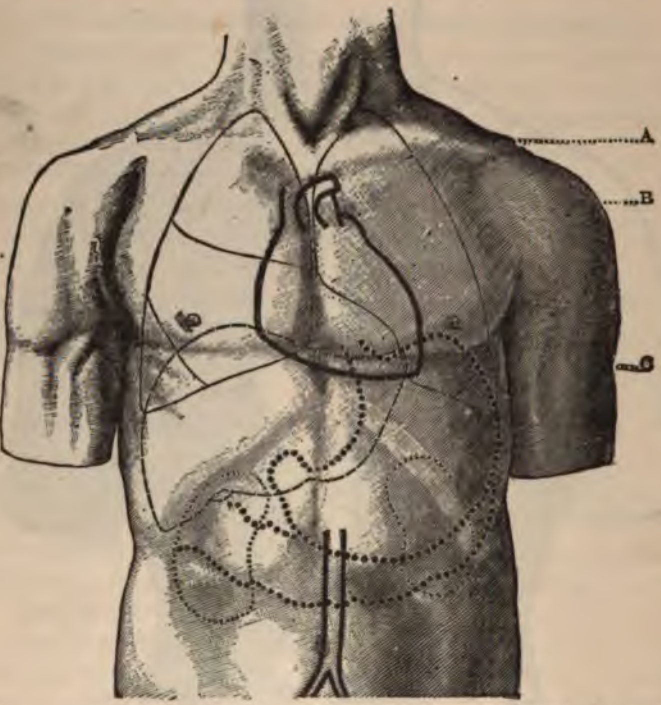

FIG. 5 (from Quain's Anatomy).—A. Upper margin of sternum = lower border of 2nd dorsal vertebra.
B. 2nd costal cartilage = 5th dorsal vertebra.
C. Infra-sternal depression (xiphi sternal articulation) = lower part of 9th dorsal vertebra.

The *movements* of the chest are of great importance in diagnosis. The free and equable expansion of the chest implies the free entry of air into the lungs; on the other hand, relative immobility or recession of any portion of the chest during inspiration, signifies that the entry of air to the corresponding portion of lung is, from some cause, retarded or impeded. In cases of general obstruction to the entry of air, whether by impediment at the main air-passage or in its entire distribution, there is universal recession of all the soft parts—the supra-clavicular regions sink downwards, the hypochondria recede, and the intercostal spaces deepen during the effort to expand the chest against atmospheric pressure. On the other hand, when the difficulty of expansion, whether from intrinsic disease of the lung or pleura, or from obstruction of air-passages, is restricted to one side of the chest or to a portion of one lung, the restrained expansion during inspiration is limited to that portion. Thus from inspection alone we may often form an opinion as to the seat, and even surmise the nature, of the disease present.

Various instruments which have already been referred to—callipers, cyrtometer, double tapes—are valuable for the purpose of *recording* differences in shape and measurement, but the information which they are useful in recording is at once obtained by the eye of the trained observer.

By *inspection* we thus learn: 1, whether a patient be large-chested or small-chested; 2, whether the shape of the chest be good and symmetrical, deformed, flattened, or bulged in any of its parts; 3, whether its movements be free and equable, or irregular and restricted, generally or locally; 4, any surface markings, enlarged veins, tumors, abnormal pulsations, etc., will at once attract the eye and be duly noted.

Palpation is employed in aid both of inspection and percussion.

(*a*) During preliminary inspection of the chest, the position of the heart's apex beat should be invariably, and as a matter of habit, ascertained, and any deviation from its normal position, viz., the fifth intercostal space one inch to the sternal side of the left nipple line, should be noted.

(*b*) Any local bulging or tumor will naturally be manipulated to ascertain its relation with bone or soft structure, whether it be solid, fluctuating or pulsating.

(*c*) In connection with percussion the observer should notice differences of resistance as well as of tone.

(*d*) Increase or diminution of *vocal vibration* or *fremitus* will be noted over any spot of altered resonance by applying the hand and making the patient utter some resonant words, such as "ninety nine."

Vocal fremitus is *increased* by consolidation of lung, provided the bronchi be not occluded; *diminished* by much thickening of the pleura, by obstruction of the main bronchus, or by air in the pleura; *annulled* by fluid in the pleura. It must be observed, however, that in some cases of fluid in the pleura a certain degree of vocal vibration may be communicated, probably from lung adherent to the chest wall above the fluid, possibly conveyed by some long bands of adhesions. The loudness or feebleness of the voice, as well as height or depth of pitch, must of course be taken into account in judging of fremitus, and corresponding part on two sides should always be compared.

(*e*) Loud coarse bronchial râles may cause the chest walls to perceptibly vibrate producing *rhonchal fremitus;* sometimes cavernous or large crackling râles will do the same. Pleuritic friction may likewise be perceptible to the hand—*friction fremitus.* In cases of effusion into the pleural cavity, or in hydatid cysts near the surface, *fluctuation* may sometimes be elicited.

Percussion is the method by which we test the resonance of various parts of the thorax and compare it with that which experience has found to obtain in health. The theory of percussion sounds is of course ultimately based upon acoustic principles; but in the multiple conditions, *quoad* percussion, present in the healthy and diseased chest, it would be impossible to give an intelligible explanation of the observed results in any moderate space, and moreover quite beyond the ability of the writer. It must suffice, then, to point out that by percussion we detect the various degrees of resonance yielded by different parts of the chest surface, which depend upon the relative amount of air and solid structure subjacent, and upon other minor causes to be incidentally noticed.

Method of Percussing.—It is best to use the fingers only for percussing. One finger of the left hand should be placed firmly upon the chest, so that the two last phalanges be accurately applied to the part percussed. With one or two of the fingers of the right hand, semi-flexed, the percussion should be made, so that the stroke fall vertically upon the applied or *pleximeter* finger. Be it observed:—

1. That the pleximeter finger be applied accurately and with sufficient firmness.

2. That it be applied precisely in the same manner and to the same spot on the corresponding sides of the chest in comparing them, *e.g.*, the finger must not be applied along the intercostal space on one side, and across the ribs on the other.

3. The percussion stroke must be made from the wrist, quite vertical to the surface percussed, and in comparing two spots the force of percussion stroke must be the same. These conditions cannot be nearly so well preserved if percussion be made from the elbow.

4. As a rule the percussion stroke should be light. The precise limits of dullness, whether in chest or abdomen, cannot be obtained by hard percussion, since the vibrations of collateral parts are too strongly elicited. It is laid down in most text-books that percussion should be made "staccato," the percussion finger not being allowed to rest upon the pleximeter. This I am convinced is an error, which is avoided by nonobservance by nine out of ten of the best manipulators in actual prac- It is sometimes, of course, advisable to employ the lightest possible

staccato percussion. It is also sometimes necessary to employ hard percussion to elicit dullness or resonance of deep-seated parts.

5. The sense of touch must be used equally with that of hearing in percussion. The degrees of resistance appreciated by the pleximeter finger are to be carefully noted. Dullness, and particularly the hardness and want of resilience over thickened and adherent pleura, may thus readily be *felt* by the pleximeter finger.

Various pleximeters and plessors are used by some observers, modifications of those designed by Piorry. The simplest pleximeter consists of a piece of ivory some two inches long and half an inch broad, the plessor being a small hammer with an india-rubber tip to the striking surface. These instruments may possibly be of value in demonstrating to a class, but their employment by students should not be encouraged for three reasons:—

1. We may rely upon having our fingers with us, but are apt to leave detached instruments behind.

2. Patients when very ill are frightened or annoyed by instruments, and may be readily hurt by them.

3. The important reason is, however, that in using such instruments we deprive ourselves altogether of the information gained by the sense of resistance. Some physicians are inclined of late to take to the pleximeter, still using the fingers as the plessor; but inasmuch as it is the pleximeter finger with which we appreciate resistance chiefly, the objection in greatest measure still holds good.

The position of the patient during physical examination of the chest by percussion and auscultation is of importance. If not in bed the sitting posture, with the back supported by a cushioned chair is the best for examining the front of the chest; if in bed, the semi-reclining posture with the back firmly supported. Whilst the back is being examined the patient should be directed to lean slightly forward and to let the arms fall loosely down between the knees, in which position the supra-scapular regions, at which the very summits of the lungs are situated, are best exposed. If the patient stoops forward too much with folded arms and bent back, the respiratory movements are impeded, and a very considerable amount of dullness may be developed at the right base by the thrusting backwards of the postero-upper surface of the liver.

Each region of the chest surface should be systematically tested by percussion, the two sides of the chest being at all points compared. Not only, however, must the two sides of the chest be thus compared from above downwards, but percussion should be employed from side to side across the sternum, so as to define the limits of the anterior margins of the lungs from either side. By this means valuable information is often elicited in cases of consumption, cancer, pleuritic effusions, etc.

TERMS.	DEFINITION.	SYNONYMS.	SIGNIFICANCE.
PALPATION.			
Vocal fremitus normal......	The transmission of laryngeal vibrations to the chest wall, appreciable by the hand.		
increased.....	....	....	Consolidation of lung.
diminished....	....	....	Bronchial obstruction or separation of lung from parietes by thickened pleura.
absent........	....	....	Effusion of fluid or air in the pleura.
Rhonchal fremitus......	Transmission of the vibration of rhonchus to the hand applied to the chest.	....	Partial obstruction of larger bronchi. Bronchitis.
Friction fremitus.....	Transmission of the vibrations of pleuritic friction.	....	Pleuritic roughening.
PERCUSSION.			
Normal resonance.....	An arbitrary term signifying the varying degrees of resonance of the different parts of the chest, within the range of health.	....	Health—needs confirmation by other signs.
Increased resonance. ...	....	Hyper-resonance.	
tympanitic resonance ..	Drum-like note........	....	Air in the pleura.
skodaic resonance ..	A peculiar form of tympanitic resonance of high pitch and great clearness.	Relaxed lung note. .	Lung in contact with surface relaxed, but not compressed by a moderate effusion into the pleura. Central consolidation in pneumonia will sometimes produce this note.
Impaired resonance....	....	Dullness of different degrees; hardness, wooden percussion.	Incomplete consolidation: co-existing *increased resistance* may often be appreciated during percussion.
amphoric resonance...	The modified resonance sometimes elicited over a cavity, and often accompanied by "cracked pot sound" (bruit de pôt fêlé).	Tubular note...........	Pulmonary excavation near the surface and freely communicating with the bronchi. Normally obtained on percussing the trachea with the glottis open.
absence of resonance.	....	Absolute dullness. Tonelessness. Flatness........ ,........	Consolidation or lung displaced by fluid or tumour.
AUSCULTATION. Breath-sounds.			
Vesicular.....	Inspiratory sound soft and breezy, expiratory sound shorter, weaker or even absent. There should be no perceptible pause between the inspiratory and expiratory sounds.	Normal breath-sounds.	Healthy lung.
Exaggerated..	Intensified normal breath-sound due to increased movement of tidal air.	Puerile. Compensatory. Supplementary........	Increased function. When heard over a portion of one lung, signifies compensatory action to make up for deficiency or disease elsewhere. Normal in young children and in adults during violent exercise of the lungs.

TERMS.	DEFINITION.	SYNONYMS.	SIGNIFICANCE.
AUSCULTATION.			
Breath-sounds (*continued*).			
Weak.........	Deficient movement of tidal air.	Feeble. Partial suppression......... ...	Diminished function.
Suppressed...	....	Absence of breath-sounds.............	Lung distant from surface or bronchus obstructed.
Interrupted...	Inspiratory sound partially or completely divided into two or three sounds.	Jerking. Wavy. Cog-wheeled............	Irregular expansion, partial consolidation about small bronchi. May be of purely nervous origin through irregular contraction of muscles.
Prolonged expiration..	Expiration lengthened to or beyond duration of inspiration.	. ...	Partial consolidation of lung or partial obstruction of bronchi.
Vesiculo-tubular.....	The vesicular part of the breath-sound being partially or completely annulled, the tubular or glottic portion of that sound heard with greater distinctness especially during expiration, which is prolonged.	Harsh. Coarse. Subtubular, Indeterminate.................	Commencing consolidation. Some authors use the term as descriptive of the roughened breath-sound of dry catarrh of the larger bronchi. (Heard normally in neighborhood of bronchi).
Tubular	A blowing breath-sound the inspiration and expiration being about equal in pitch and duration and distinctly divided. Placing mouth in position to pronounce word commencing with guttural *ch* (χ) and drawing breath to and fro imitates the sound with exactness (Skoda).	Bronchial. Blowing. Tracheal. May be high pitched or whiffing, medium or low pitched...	Hepatization or consolidation of the lung. (Heard typically over trachea.)
Carvernous...	A blowing breath-sound of hollow quality, most so in the expiratory portion, which is usually of lower pitch than the inspiratory.	....	Pulmonary excavation or condensed lung with dilated bronchus.
Amphoric.....	Similar to the above, but with blowing character and hollowness intensified.	....	Large pulmonary cavity.
Adventitious sounds.			
Rhonchi	Musical sounds generated by partial obstruction in a bronchial tube imparting vibrations to the air currents.	Râle (râle and rhonchus being indifferently used by many authors).	
Sonorous rhonchi...	Low pitched, loud snoring.	Sonorous râle.........	Bronchitis of the larger tubes.
Sibilant.....	High pitched, whistling	Sibilus	Bronchitis or spasmodic narrowing of the medium or fine tubes.

Terms.	Definition.	Synonyms.	Significance.
Adventitious sounds. (*continued*)			
Stridor.....	A coarse vibrating rhonchus, generated at the larynx or by pressure on the main bronchi.	Stridulous rhonchus or râle..................	Pressure of a malignant or aneurysmal tumor upon a main bronchus or the trachea, Sometimes produced by laryngeal paralysis.
Râles..........	Moist sounds or rattles produced by the bubbling of air through fluid in the lung or bronchi.	"Rhonchi "and" râles" are used indifferently by many authors.	
Small crackling	A fine râle produced in the minute bronchioles and alveoli of consolidated lung consisting of numerous small sharply defined crackles chiefly audible during inspiration, but in less degree with expiration.	Subcrepitant râles Moist crepitations...	Thin fluid in minute bronchial tubes with consolidation of the lung. Resolving pneumonia.
Medium crackling.	Similar to above of larger size.	Crepitant râles........	Resolving pneumonia. Broncho-pneumonia. Rapid caseous pneumonic softening.
Large crackling.	Crackles of larger size and fewer in number produced in minute pulmonary cavities.	Moist crackling........	Softening pneumonia or tubercle.
Gurgling....	Larger and more liquid râles produced in cavities of medium and large size.	Cavernous râle.........	Cavity in the lung.
Small bubbling......	A râle produced by the bubbling of air through mucus in the finer bronchi, and more or less muffled by transmission through spongy lung.	Sub-mucous or muco-crepitant râle........	Capillary bronchitis. Pulmonary œdema
Medium and large bubbling......	Similar râles generated in larger tubes.	Mucous râles. Tracheal rattles......	Bronchitis of larger tubes. Secretion collecting in the trachea during last moments of life.
Clicking sounds	Single sounds, or few in number, mostly limited to inspiration, and of sticky, semi-fluid character.	Dry crackle............	Commencing softening of tubercular deposits in the lung.
Crepitation...	A minute dry crackling sound in which the crackles are infinitely small and even, and occupy chiefly the latter part of inspirations.	Fine dry crepitation. Pneumonic crepitation.	Early stage of pneumonia. The sound is sometimes heard in a certain degree of pulmonary œdema, and during the first two or three deep inspirations over a portion of lung not used (*e.g.* the extreme bases in bedridden patients).
Metallic tinkling.........	The metallic resonance sometimes imparted to a moist sound by a large pulmonary or other cavity. The sound may be generated in the cavity or resonated from a bronchus in connection with it.	....	A large dense walled pulmonary cavity or a pleuritic cavity.

TERMS.	DEFINITION.	SYNONYMS.	SIGNIFICANCE.
Adventitious sounds (*continued*).			
Splash	The succussion of air and fluid produced by the shock of cough in a large cavity or by shaking a patient, with the ear applied over a hydro-pneumo-thorax.	Hippocratic succussion sound. Cough splash.	A large cavity containing air and fluid. Hydro or pyo-pneumo-thorax.
Bell sound	A metallic ring heard on sharp percussion over a pneumo-thorax, commonly elicited by the use of coins.	*Bruit d'airain*	Pneumo-thorax.
Friction	A rubbing sound produced by the movements of two surfaces of the pleura that are in contact, and inflamed or roughened.		Pleurisy.
Dry Friction		Leathery or creaking friction. Dry rub.	Heard at commencement and at termination of pleuritic attack.
Moist friction	A sound often closely imitating moist crepitation produced by attrition of surfaces covered by soft moist lymph.	Spongy friction. Friction crepitus, etc.	Sometimes heard over upper confines of recedent effusion and in other conditions of thick moist pleuritic exudations.
VOICE SOUNDS			
Normal	The sound of the voice transmitted through the healthy lung.		
Increased or diminished.			
Annulled or absent			Lung separated from chest wall by fluid or growth.
Bronchophony	The loud transmission of the laryngeal vibrations, apart from articulation.		Pulmonary consolidation.
Pectoriliquy	The clear transmission of articulate sounds. Heard best during whispering, when bronchophony (which usually, but not always accompanies it) is excluded.		Cavity in the lung. A close imitation of this sound *pectoriloquie aphonique* may be sometimes heard through a pleuritic effusion (sero-fibrinous) on making the patient whisper roughly.
Ægophony	High pitched, tremulous modification of the voice, due to the transmission of the upper tones or harmonics.		Pleuritic effusion.

The foregoing table includes all the terms used in a technical sense, which are necessary for describing the sounds met with in chest diseases. They are—with one or two unimportant additions—those which were carefully selected by my friend Dr. Mahomed and myself, as English members of a committee nominated at the International Medical Congress held in London in 1881, to endeavor to simplify the terminology of auscultation for international use. The list has at least the advantage of not including any new or unknown terms, and as it only differs on the side of greater simplicity from the terminology in the last edition of my work, and in my more recent article on physical signs in Quain's *Dictionary of Medicine*, I venture to introduce it here pending the final report of the committee above mentioned.

A few observations are still necessary upon the mechanism of chest signs. The results of clinical observation will not bear out the views current since the time of Laennec with regard to many points in the mechanism of auscultatory phenomena—views, however, which have not escaped the criticism and opposition of such able observers as Beau, Spittal, Skoda, Bondet, Chauveau, and others. Our difficulty indeed lies in arriving at a true judgment amidst the conflicting and ably maintained opinions of many authors, in gathering and knitting together the fragments of truth which lie scattered over their extended battle ground. I will endeavor as briefly as possible, and in the way of comment upon some of the headings of the above table, to give the rationâle of the physical signs there mentioned.

PERCUSSION.—The explanation of the sounds elicited by percussion is comparatively simple. The degrees of resonance and impaired resonance are dependent upon (1) the proportion of air and solid under percussion, (2) the tension of the air within the chest cavity, (3) the range of vibration of the chest wall.

In the well-formed chest of the young adult, and within the range of normal conditions of quiet breathing, we obtain over the pulmonary regions that degree of resonance or clearness of percussion which is typical of health. At any stage of the inspiratory act, however, the percussion note is somewhat deadened by closure of the glottis and compression of the chest by the expiratory muscles, the tension of the confined air being increased and its vibratility and that of the chest wall diminished. It is said that the chest resonance is increased at the end of deep inspiration. This is only true about the confines of normal resonance. On trial it will be found that the clearest percussion note is elicited within the range of calm breathing. In pneumo-thorax, with free communication with a bronchus, *i.e.*, when the air has approximately the normal atmospheric tension, we get the most deep-toned percussion resonance (tympanitic resonance). If, however, the communication with the lung be only by a valvular opening admitting of entry, but not of escape of air, we get increased tension and correspondingly deadened note, as the air accumulates with distension of the chest wall. Dr. C. J. B. Williams[1] and more recently Dr. Bristowe[2] both regard the percussion note as primarily due to the vibration of the chest wall and as modified by the conditions of the underlying cavities or viscera, so far as they afford varying degrees of impediment to the chest-wall vibration. In the above-described circumstances it is true we cannot separate increased tension of air from increased tension or rigidity of solids, and there are many states in disease, and even within the range of health, in which the resilience of the chest walls is impaired, and an impairment of resonance thereby accounted for. Thus many robust people, soldiers especially, have rigid resisting chest walls, and percussion in such persons yields a diminished sound. On the other hand in children, and in delicate persons with thin elastic ribs, we may get an amount of resonance which may even mask a certain degree of underlying disease. Mere adhesion of the pleural surface, *i.e.*, the delicate spongy adhesion without appreciable thickening, is not sufficient to alter percussion note, since it does not of itself impair mobility. A thickened adherent pleura gives rise to a dead, hard percussion note. Underlying cavities or dilated bronchi will give certain degrees of tubular or amphoric resonance, especially whilst the glottis is open, but the resonance in such cases is always less full than, as well as different in quality from, that of healthy lung. In percussion, it must be borne in mind that over a surface where, from the underlying conditions, we should expect complete dullness, we may yet elicit a considerable degree of resonance conducted from adjacent parts containing air. This lateral conduction from resonant areas, modifying the percussion note over areas of consolidation, is more noticeable on hard than on light percussion, and it is only by gentle percussion that the limits of dullness can be accurately defined.

In the healthy chest we obtain, during moderate expiration, *superficial cardiac dullness*, corresponding with that small area marked off by a horizontal line drawn from the apex beat to the mid sternum, and from the same point (the apex beat) to the lower margin of the fourth cartilage at its junction with the sternum. On firm percussion, the *deep heart's dullness* may be elicited as high as the third cartilage, mid-way between the nipple and the sternum, and to the right margin of the sternum at the nipple level. *Stomach note* is obtained at the sixth cartilage in the left mid sterno-nipple line; in the left posterior axillary line a small area of dullness, extending upwards from the margin of the ribs for a couple of inches, marks the situation of the *spleen*. On the right front of the chest in the nipple line, *liver dullness* is obtained below the level of the sixth rib, and on deep percussion for a rib higher; posteriorly, about two fingers' breadth of dullness at the extreme right base marks the situation of the liver in contact with the thorax. Lung resonance is obtained anteriorly over the supra-clavicular fossa, and posteriorly extends upwards to the level of the seventh cervical spine, *i.e.*, about 1½ in. above the level of the clavicle. It is in these highest regions of the chest, corresponding with the extreme summits of the lungs, that the first evidence of phthisical disease is often obtained.

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[1] Diseases of the Chest, 4th edition, p. 13.

[2] Practice of Medicine, 5th edition, p. 372.

Auscultation.—With regard to respiratory murmurs or breath-sounds it may be broadly stated that all breath-sounds, whether healthy or morbid, with the exception of normal vesicular inspiration are generated in the larynx, and modified in their transmission through the different media normally, or abnormally, intervening between the larynx and the point of observation.

The following propositions express, I believe, the exact truths respecting the nature of breath-sounds (excluding râles and adventitious sounds).

1. The vesicular respiratory murmur is a sound having a double mechanism being made up of (*a*) a bruit produced by the passage of air to and fro through the glottis, and reverberated downwards through the bronchial tubes, (*b*) an infinite number of minor bruits similarly produced at the openings of the pulmonary lobuli.

2. All other breath-sounds (excluding adventitious sounds) are due to the conduction of the glottic sound above mentioned through media of different kinds, forms, and densities; any local currents of air being only so far operative in as much as they serve to assist conduction.

3. It may be observed that, although in cavernous and amphoric breathing the breath-sounds may be generated locally by the air currents entering and issuing from the cavity, yet this local mechanism is not essential to the sounds, and is often wanting.

Vesicular murmurs.—It would seem a simple thing to demonstrate once for all the production of vesicular murmur. Yet in truth the conditions of its mechanism are most difficult to reproduce. If the lungs, remaining in the chest, or placed upon the table, be auscultated, whilst air is injected through the trachea, sounds are doubtless heard more or less closely resembling those of vesicular breathing, but in such an experiment the whole mechanism of respiration is inverted. In natural breathing, the air is not injected into the lungs—it enters to fill the space that would otherwise be left vacant by their expansion; the important difference being, that in inspiration there is no period at which any air tension beyond that normal to the atmosphere exists. The lungs are never inflated, nor are they ever even approximately emptied of air, so that in truth the function of respiration proceeds by interchange of gases within an area of comparative calm, only so far disturbed by tidal and secondary currents as to effect its thorough ventilation. But at the narrow isthmus of the glottis through which the air—small in amount compared with the lung capacity—has to pass and repass sixteen or seventeen times a minute, the current is rapid, and the air necessarily cast into audible vibrations, producing a blowing sound which, carried along the bronchial channels, becomes infinitely divided, hushed and broken up, to form part of the rustling murmur of normal inspiration. M. Beau,[1] in 1834, first sought to prove that tracheal, vesicular, bronchial and cavernous breath-sounds were not due, as Laennec had thought, to the friction of the air against the parietes of the trachea, alveoli and caverns, but that they resulted in the manner above mentioned, from the reverberation in these parts of a single sound which was produced in the superior air passages.

MM. Barth and Roger observe, however, that the inspiration loses nothing in force at the base of the lung, and maintain that the vesicular murmur ought consequently to have its origin in part, at least, in the lungs themselves. And indeed my own observation goes to the support of their opinion—viz., that the respiratory murmur in the majority of instances, does not lose intensity towards the base of the lung. Even if it be so, however, I do not see how the fact should invalidate M. Beau's view, for the propagation of a sound in the bronchial tubes—which remain open through the traction exercised upon them by the elastic lungs—is too perfect to suffer any diminution in a distance so small as that which separates the summit from the base of the lung.

On the other hand, M. Beau repeats the observation of Laennec that, in animals with long necks, such as the ruminants, the respiratory murmur is much more feeble than in the carnivora, as evidence in favor of the glottic production of the respiratory bruit. One has possibly in such case, however, to deal with differences of structure and of functional activity.

Dr. Spittal,[2] writing but a few years after Beau's views were first published, brought forward some additional experiments in support of them, in which by eliminating the respiratory movement of the lungs as a factor, he sought to show how far the respiratory sounds were really conducted laryngeal sounds.

Skoda also believes the respiratory murmur to be due to a double cause, viz., the conduction of the glottic bruit through the bronchial tubes, and a sound produced in the air-cells and small bronchi. During expiration, the air passing from a large space, the air-cells, into a smaller, the bronchi, becomes compressed, and hence the expiratory murmur of the larynx, trachea and large bronchi is as a rule louder than the inspiratory. Dr. Blakiston,[3] who attributes the pulmonary sound to the air rushing through the smaller bronchial tubes, well points out that there is no contractile resistance within the lung to be overcome by the entering air, the lungs being expanded not by the injection into them of air, but by the enlargement of the thorax by the inspiratory muscles. And it is curious to note that Skoda himself when criticising a statement of Fournet's, implying a dilating power of the air entering the lungs, remarks, "Air enters only when a vacuum is formed, and does not struggle against obstacles, except when a vacuum exists beyond these."

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[1] Archives générales de médecine, 1834, 2me série, tom. v., p. 557.

[2] On the cause of the sounds of Respiration. Edinburgh Medical and Surgical Journal, vol. li., 1839.

[3] Diseases of the Chest, p. 18.

I must not conclude the review of the chief opinions that have been held respecting the mechanism of the respiratory murmurs without referring to a most important, and to my mind the only conclusive experiment that has been recorded, correcting the too exclusive theory of Beau. The experiment was conducted by MM. Bondet and Chauveau[1] upon a horse suffering from pneumonia affecting the lower half of the left lung. On auscultating the animal, exaggerated breathing was heard over the right lung and over the upper half of the left, tubular breath-sound being distinct over the lower or affected half of the left lung. These preliminary observations made, the trachea was opened by an incision twenty centimeters in length, and the following phenomena were noted:—

(1) On auscultation over the trachea below the incision, the wound being held widely open, the inspiratory bruit was almost completely lost, and the expiratory sound but faintly audible.

(2) On auscultation over the consolidated lung, whilst the wound in the trachea was held widely open, no tubular sound could be heard during inspiration, and only a *faint, brief and abortive sound during expiration.* Over the rest of the lung and over the opposite (sound) lung, however, the *normal respiratory sounds were clearly heard with quite normal distinctness.*

(3) A tube supplied with a musical reed being introduced into the trachea, the artificial voice-sound was heard very distinctly over the consolidation, but was inaudible over the rest of the lung.

This experiment at first sight appears almost conclusively to show that the whole of the respiratory murmur is generated within the lung, since it was not notably impaired by eliminating the glottic element. The artificial voice-sounds, on the other hand, audible over the consolidation, showed that a possible fallacy, viz., the blocking of the left lower bronchi by blood clot, did not exist.

The glottic bruit is, however, a fact which cannot be denied, and that the sound is propagated downwards to the lung is equally certain from the experiments of Beau, Spittal, Barth and Roger, some of which I have myself repeated; nor is it possible as a matter of reasoning to conceive, but that a sound, loud in the trachea and traceable down the bronchi so far as they can be reached, should enter as an element at least into deeper sounds. I have also already directed attention to the tense condition of patency, in which the walls of the bronchi, the minute bronchi especially, are held by the elastic tissue of the lung in which they are imbedded, as being highly favorable to the reverberation of sound through them. I must confess to great doubts whether the movement of the air in the bronchi gives rise to any appreciable sound, and whether even the angles of subdivision of these tubes would suffice to throw the tidal air into audible vibrations. It can scarcely be doubted that the intra-pulmonary portion of the inspiratory murmur is generated, as suggested by Bondet and Chauveau, at the junction of the bronchioles with their infundibula. Here are repeated on a minute scale, but at innumerable points, the conditions of a vibratile aperture terminating with considerable abruptness in an expanded chamber, conditions which are favorable to the production of *veines fluides* whose sum total amounts to a considerable sound of such a kind as that of vesicular inspiration.

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[1] Revue mensuelle de médecine et de chirurgie, tome i., 1877, p. 161.

All experiments seem to show that the expiratory murmur is of extra-pulmonary, *i.e.*, glottic, origin.

Weak, harsh (vesiculo-tubular), and *exaggerated breathing.*—Weakness of breath-sound may simply arise from feebleness of the muscular chest movements; it is then better spoken of as partially suppressed breathing. When the weakness of breath-sound arises from defect in the lungs it is always accompanied by some harshness, the true vesicular sound of the lung being more or less completely wanting and the glottic sounds faintly audible, their conduction being partially obscured by occlusion of some of the finer bronchi. In *harsh* breathing the glottic sounds are better conveyed to the ear by the partially consolidated lung. In this form of breath-sound the expiration is always more audible and prolonged than natural, and the inspiratory and expiratory sounds are distinctly divided by a very brief interval, this breath-sound being in fact the tracheal sounds conducted from some distance. There is a kind of "harsh" breathing which would, however, be better styled rough or coarse dry vesicular breathing, which is commonly heard in the neighborhood of small and cicatricial deposits. This sound is probably a true pulmonary sound, it resembles coarse and exaggerated inspiration of very dry character; which features are not distinct in the expiration.

Exaggerated breath-sound is heard in the healthy young child. More rapid breathing, more rapid passage of air through the glottis and into the lungs, are the conditions upon which this enhanced breath-sound depends. The expiration is generally rather prolonged, and both sounds are coarser than ordinary. Exaggerated breath-sound is usually, in health, limited to children of tender years, and hence its synonym "puerile breathing," but some persons preserve this quality through adult life. This variety of breathing is heard over the sound side in cases in which the respiratory function of one lung is in abeyance from any cause. It may be similarly heard over one portion of a lung, the rest being diseased. Hence another synonym, *compensatory breath-sound*. It indicates respiratory vigor and is so far of good augury, inasmuch as it shows that an enlarged lung, or portion of a lung, is not merely dilated but also functionally more active, a most important fact to ascertain in cases of one-sided chest disease.

When a lung is separated from the surface by an interval of fluid or has its bronchus occluded from any cause, we get *suppression of the breath-sounds* over it. In order for this suppression to be complete in pleuritic effusion, the effusion must be sufficient to compress the lung; otherwise feeble bronchial breath-sound is conducted through the fluid. Even in cases of considerable effusion, however, a certain amount of breath-sound is heard over the upper part of the chest, although it is obvious that the collapsed lung can take no part in producing the sound. When the lung is separated from the thoracic wall by greatly thickened adhesions, and —as is usually the case—is itself indurated and collapsed by interstitial growth, the breath-sound is partially suppressed, the glottic bruits being only very imperfectly conducted.

Jerking or *wavy* and *cogged* breathing are varieties to which different authors attach very different importance. In jerking or wavy breathing the inspiration, instead of being a continuous sound, is two or three times interrupted. The expiration is rarely affected in this way, and may be normal or simply harsh and somewhat prolonged. One so frequently meets with jerking respiration in nervous people, that when unaccompanied by other morbid sounds but little importance is to be attached to it. It may be due to, (*a*) irregular action of the respiratory muscles under conditions of pain, as in pleuro-dynia pleurisy, myalgia, and also in hysteria and other nerve disorders; (*b*) (when on the left side) to cardiac pulsation, it being then of strictly cardiac rhythm; or (*c*) it is possible that in nervous people during excitement of the heart's action, the impulsion of blood through the pulmonary small vessels and capillaries may cause some sensible modification of the respiratory rhythm; (*d*) certain textural diseases of the lung itself, causing a want of uniformity in elasticity and expansile power, may account for some cases of local waviness of breath-sound (Dr. Walshe considers that pleuritic adhesions may do so); (*e*) waviness of breathing caused by any of the above circumstances may be superadded to other more definite signs of lung disease. Thus pulsatile waviness of cardiac rhythm is often combined with well-marked cavernous respiration. A remarkable instance of wavy, cavernous breathing was witnessed by the author in a case in which the entering bronchus to a large cavity was partially occluded by a small pulmonary aneurysm projecting into its calibre close to the cavity, and thus alternately stopping and permitting the cavernous sound. The death of the patient from sudden hæmoptysis within a few hours verified and explained this observation.[1]

Cogged wheel rhythm is a character of the respiration to which much importance is attributed by some authors. It is attributed to minute obstruction in the finer bronchial tubes, probably by glutinous and adherent mucus (Walshe), which the current of air succeeds in forcing at intervals without producing any distinct bubbling. Dr. Walshe believes that this sound is due to the same cause as liquid râles, into which it is finally converted. This sound has some affinities with wavy respiration, and, like it, is commonly limited to inspiration, but the inspiratory sound is more subdivided.

Prolonged expiration.—Prolongation of expiration is perhaps the most important, as it certainly is the earliest of the signs of commencing consolidation of the lung. It marks the transition between vesicular and bronchial breath-sound, and it indicates that over a certain extent of lung the vesicular element of breath-sound is diminished or lost, glottic sounds becoming predominant, or alone heard, with their distinctive characters and proportion. Thus with prolonged expiration we get division between inspiration and expiration (*divided respiration*), a characteristic of the glottic type. The vesicular quality of the inspiration is gradually lost as the expiration becomes more distinct, *i.e.*, harshness is always combined with prolonged expiration. The expiration instead of being inaudible, or occupying but a fractional part of the duration of inspiration, comes to equal it or even slightly to exceed it in duration. These are *par excellence* the characteristics of the glottic breath-sound, and as, on the one hand, prolongation of the expiration-sound is associated with harshness, *i.e.*, want of vesicularity of the respiration, so, on the other, it passes into true bronchial breathing, in which the glottic sounds are conducted in all their intensity. I may here remark that in listening for characteristic moist sounds or râles, our attention will be attracted mainly to the *in*spiratory portion of respiration, during which most of the morbid sounds are heard; whilst modified breath-sounds are most characteristically heard during *ex*piration.

Bronchial respiration.—This variety of respiratory murmur is heard normally at the lower part of the trachea, and in less degree at the interscapular region at the level of the spinous process of the fourth dorsal vertebra; as a morbid sign it is best heard over a lung consolidated by acute pneumonia. Skoda pointed out that this breath-sound could be best imitated by placing the tongue in the position to form the consonant *ch* (hard), χ, and drawing the breath to and fro.

Bronchial breathing varies in intensity according to the degree of consolidation, the proximity to the main bronchial channels, and the condition of the secondary and tertiary bronchial divisions. Most auscultators think with Laennec that the sounds are originated in the affected lung; but on reflection one is compelled to regard it as a sound conducted from the larynx for the following reasons:

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[1] Path. Soc. Trans., vol. xxii., p. 48.

1. In pneumonia, in which it is best heard, the lung is injected by a coagulated substance which fills it, and maintains it in the state of permanent inspiration, a condition which forbids the entry of air. There no longer exist the currents of air in the bronchi of the affected lung. MM. Barth and Roger maintain, and with them my friend and colleague Dr. R. Thompson agrees, that the inspiratory air-current passing to inflate the sound lung secures the entry also of air to the diseased side. Any air so obtaining entrance, however, could scarcely produce a respiratory sound.

2. It is not certain that the air moving in the bronchial tubes of itself produces any sound. Probably such air currents serve but to facilitate the propagation of vibrations which originate elsewhere.

3. The experiment of Bondet and Chauveau upon the hepatized lung of the horse already given (p. 34), appears to me to settle the question. These observers on holding widely open the wound in the trachea caused the respiratory bruit heard over the hepatized lung to disappear, whilst over the sound lung the respiratory murmur was scarcely at all modified.

It is probable that the bronchial tubes vibrate in unison with the air which they contain, and thus augment the sounds which they convey. It would appear then, that the two conditions for the production of bronchial breath-sound are consolidation of lung and patency of bronchi. Any obstruction of the main bronchus will obscure the sound, and the separation of the lung from the parietes, whether by fluid or by thickened pleura, renders it less distinct. In some rare cases of pneumonia the breath-sound is suppressed, I presume from the small bronchi being occupied by exudation overflowing into them from the alveoli. Bronchial breath-sound may be produced by other than lung conditions. A mediastinal tumor, aneurysmal or cancerous, situated between the thoracic wall and a bronchus will yield the sound.

The expressions blowing, tubular and tracheal, merely have reference to extent and intensity of bronchial breathing, *blowing* referring rather to extension than intensity, this degree of bronchial breathing being heard when the lung is incompletely consolidated; *tubular* breathing being the most characteristic quality of bronchial breathing, *tracheal* respiration still more intense, being heard over consolidation in the neighborhood of the main bronchi or their primary divisions.

Cavernous breath-sound.—This consist of a hollow inspiratory and expiratory sound, which has been compared to the sound produced by blowing into the cavity formed by the two hands. The expiration must be especially attended to in listening for cavernous breath-sound; it is wavering, hollow and prolonged. Drs. Flint and R. Thompson, both attach much importance to the fact that the expiration is of lower pitch than the inspiration, as serving to distinguish cavernous respiration from bronchial, where the inspiratory and expiratory murmurs are of similar pitch. Cavernous respiration is heard over pulmonary cavities, (1) which are of a certain size (larger than a filbert), (2) which are in part at least empty, (3) which communicate freely with one or more bronchi. As to the mechanism of this sound, one cannot deny that it may be imitated by breathing into the hollow of the two hands, but it is questionable whether this mechanism suffices to explain the cavernous respiration heard over the lungs.

1. In the first place, the cavernous sounds are heard during the whole period of inspiration and of expiration. Now I doubt the possibility—but no one would deny the rarity—of the existence of a cavity in the lung capable of contracting and expanding with such completeness as to generate the sounds: for the air will only enter such a cavity (except when impelled there by the act of coughing) in accordance with precedent expansion.

2. One again and again meets with cases in which very well-marked cavernous breathing may be heard over large cavities whose powers of expansion are almost *nil.*

3. The observations of Beau, Spittal and Skoda lead them to adopt this view respecting cavernous sounds in preference to that of Laennec more usually accepted. It is doubtful, however, how far "consonance" in the right sense of that term is concerned, as Skoda maintains, with the production or reinforcement of the cavernous sounds. For there can be no definite relationship between the bronchial and cavern spaces and their walls which would enable them to attune to one another save in a very haphazard way.

Amphoric breathing.—This is really a variety of cavernous breathing, the cavity being large, superficial and thin walled.

There can be no doubt, however, that in some cases, in which the cavity is large and the thickened tuberculous lung textures so removed as no longer to restrain the chest movements, direct influx and efflux of air through the bronchial tubes communicating with the cavity will cause the sound. In cases of pneumo-thorax, in which the opening is free, amphoric breath-sound is heard to perfection; if the communication be valvular the sound is not heard at all, or very imperfectly so.

Râles.—With regard to the different rhonchi and râles, some are of such obviously simple mechanism as to require but little explanatory comment. The rhonchi, for instance, are but musical notes of higher or lower pitch, depending upon vibrations of air set up in tubes, partially obstructed by local thickenings of their lining membranes, by mucous collections or pellets adhering to their sides so as partially to obstruct them, or finally by spasmodic narrowing of the tubes. It will be observed that all these causes of sibilant and sonorous rhonchi within the chest are of a temporary or transient nature, and accordingly these sounds are very inconstant, appearing and disappearing, or shifting from one spot to another, even whilst the patient is under examination. A vigorous cough, clearing the tubes of mucous collections, will commonly for a time remove a sonorous rhonchus, and the sibilant sounds in asthma or bronchitis with spasm, will be partially or wholly removed by the inhalation of chloroform or ether. It must further be noted that in all cases the true respiratory murmur is obscured and enfeebled by the presence of these rhonchi, to return on their removal.

Stridor is a variety of the sonorous rhonchus in which the vibrations are very coarse, and the sound produced very hoarse and low pitched. It may be occasioned by some paralytic affection of the glottis, which prevents the cords from being drawn apart during inspiration, and allows them partially to flap together, and thus to vibrate. It is in other cases produced by the pressure of a tumor directly upon a main tube, as the trachea or main bronchus. The vibrations which produce stridor, and those of the stronger forms of rhonchus, are so coarse as to be perceptible to the hand applied to the surface of the chest, thus producing one kind of *rhonchal fremitis.* It may be noted that the sounds under consideration are chiefly, but by no means solely, heard during inspiration, the coarser forms and especially stridor being more particularly limited to that portion of the breath-sound.

Great difference of opinion has prevailed respecting the nature and mechanism of *crepitant râle* or fine crepitation. All observers agree that it is most typically heard in the first stage of pneumonia, and some regard it as the pathognomonic sign of that disease. This sound is defined by Walshe as composed of a variable, sometimes immense, number of sharp crackling sounds of minute size, all perfectly similar to each other, and rapidly evolved in puffs more or less prolonged. The sounds are dry in quality and co-exist exclusively, except in rare cases, with inspiration; and once established remain a persistent condition, until superseded by other phenomena. Williams's description of the sound, as resembling that produced by slowly and firmly rubbing a lock of hair between the finger and thumb close to the ear, has been generally accepted as an accurate one. The fine crepitation of pneumonia is very exactly imitated by other conditions: pulmonary collapse from disease, certain degrees of pulmonary œdema, œdematous pleura, and the first stage of pulmonary apoplexy, may be instanced as several circumstances in which the sound may be produced, and the only condition common to all cases is the expansion of a moist and heterogeneous tissue. The application of the term *dry* to the sound, although acoustically correct, is peculiarly misleading, for moisture of tissue is probably one of the conditions essential to its production. Thus the sound may sometimes be most typically heard over a pleura, the two layers of which are separated by œdematous cellular tissue, a condition commonly found over old contractile lung consolidations. Again, in œdema of the lung, as for instance in some forms of heart disease and albuminuria, the fine crepitation may be well heard. In saying however, that fine crepitation may be well heard under these several conditions, I am bound to confess that there is to the fine crepitation of pneumonia an element superadded, a peculiar quality of the breath-sound, due to the consolidation of the lung, something short of the typical bronchial quality, and yet far beyond mere harshness, which gives a special character to the crepitant râle, and increases its loudness and definition. The fine crepitation under other conditions is, as a rule, unattended with any breath-sound. The actual state of lung in pneumonia which gives rise to the crepitation may, I think, safely be assumed to be one of œdematous exudation from congestion. When the exudation into the air cells is complete and consolidated, the crepitation ceases.

Sub-crepitant râle is a small bubbling râle audible during both inspiration and expiration, but chiefly so with inspiration, generally occupying the beginning only of the expiratory sound. It is typically heard in the resolution stage of pneumonia, at the period when the sputa are becoming opaque and more or less purulent. This râle is more commonly heard than the dry crepitation in the moist catarrhal pneumonia which complicates phthisis. In œdema of lung beyond mere wetness of tissue, in which the fine bronchial tubes at the bases become partially occupied by thin fluid, although still permeable to air, the râle is also heard. Its mechanism consists simply in the presence of this fluid in the fine bronchial tubes, and it is most typically heard in those cases in which, as in pneumonia, the bronchial tubes are surrounded by consolidated lung. The transitions between sub-crepitant and sub-mucous, and mucous râle are very fine, and, minutely regarded, are of no practical importance. We have often to appeal to other signs, the character of the respiration and the presence of percussion resonance or dullness, to help us in the interpretation of these râles. Thus, in capillary bronchitis of the adult, we have a fine bubbling râle of comparatively muffled character, which is attributable to secretion in the small bronchial tubes, but the same condition in the infant—capillary bronchitis without any lung involvement—will give rise to a very sharply defined, almost metallic sub-crepitant râle, not to be distinguished from that of broncho-pneumonia or pneumonia undergoing resolution. The absence of bronchial breathing or of dullness will in some cases help us in the diagnosis, in others we must rely upon the general features of the case.

Dry crackling is a sound comparatively rarely heard. It consists of a few, usually two or three only, sharply defined clicks heard during the latter portion of a deep inspiration; the inspiratory murmur being enfeebled or absent, and the expiratory sound prolonged, and more or less bronchial. The sound is said to be significant of commencing softening of "tubercular" deposits. It is also heard over centres of inspissating morbid products. In the former case a crepitant râle, and in the latter a moist and more abundant crackling râle, is delevoped by cough in many instances.

Humid crackling, moist crackling, humid clicking, or by whatever other synonymous phrase we designate it, is a sound very frequently heard in the so-called second or softening stage of phthisis, and in the adult the sound is very significant of pulmonary softening; indeed the true humid clicking means commencing destruction of lung, and is usually associated with pulmonary tissue in the sputa, a sharply defined, moist or bubbling sound consisting of two or three inspiratory and one or two expiratory elements. The respiratory murmur is masked or replaced by the crackles, and does not return until, by enlargement or coalescence of the minute cavities giving rise to them, the râles have become enlarged to cavernous râles, or gurgling, and the breath-sound has become blowing or cavernous in quality. The humid crackle varies much in degree or sharpness of definition according to the condition of the pulmonary tissue situated between the centres to whose softening it is due. If these centres are but necrotizing islets scattered through a large tract of lung, all of which is consolidated by catarrhal pneumonia (or mixed catarrhal and croupous pneumonia), as in the acutest form of pneumonic phthisis, the clicks are very strongly conducted, very metallic, and mingled with other coarse crepitant sounds. But in other cases the softening nodules are more or less widely separated by spongy and tolerably healthy lung tissue, when the humid clicks are muffled and more or less obscured. Over portions of lung which whilst undergoing softening are, owing to fibroid change or pleural thickening, functionally inactive under ordinary circumstances of respiration, no moist sounds may be heard until a sharp cough has forced air into the softening tissues and developed the sounds. This is a most important point to remember in auscultation.

Cavernous rale—gurgling rale. With enlargement or coalescence of small cavities to the size of, or exceeding a walnut, a second possible mechanism is to be taken into calculation as accounting for the large liquid râles produced.

Of course fluid bubbling in a cavity will account for the sounds heard, as we may perceive on listening over a secreting cavity whilst the patient coughs, when we hear the splashing and gurgling sounds from forcible commingling of air and fluid. But in large and comparatively dry cavities we can hardly imagine any such commingling of air and fluid to take place, during ordinary or deep breathing; even supposing, as I have endeavored to show is not necessarily the case, air to pass to and fro into the cavity with any appreciable current. Any mucous râles produced in the associated bronchi, will, however, be heard with exaggerated intensity over the cavity, and if the cavity be of sufficient dimensions will give rise to metallic tinkling sounds. It is entirely erroneous to suppose—as may be demonstrated frequently on *post-mortem* inspections—that metallic tinkling is significant of pneumo-thorax only: it may be most typically heard over a large cavity, and its mechanism is probably the same in both instances, viz.: the reverberation through a large thin-walled and empty cavity of a moist râle produced in the bronchus, or fistulous canal leading to the cavity.

Voice-sounds.—Enough has beeen said in the earlier part of this chapter respecting the mechanism of breath-sounds to make it clear, that, to my mind at least, there can be no good reason for any mystery, as to the way in which voice-sounds are intensified or annulled under different morbid conditions of the lungs and pleuræ. There can, at all events, be no doubt that voice-sounds are conducted from the larynx, and the more the expiratory sound is broken up into articulate sounds the more favorable are the conditions for transmission backwards through the bronchial channels. These channels in the normal state are, as already stated, held patent by the elastic traction upon them of the surrounding lung, and they serve therefore as so many speaking tubes, although the direction of the current of air during articulation is not favorable to the best possible conduction.

The rapid subdivisions of the tube and their being imbedded in an ill-conducting material, is, however, highly unfavorable to conduction of sound, and were it not for the shortness of distance the voice sounds would not be audible at all. Up to the point of subdivision of the main bronchi, a few inches more or less of tubing would make but little difference in intensity of conduction; hence it is that mere linear distance from the glottis does not tell much upon the conduction of voice-sounds. They are in health pretty nearly as well or as badly heard at the base as at the apex or middle of the lung. If the glottis be destroyed by tubercular or syphilitic disease the voice-sound is of course annulled, and even the whisper sound is impaired, showing that the glottis is to some extent concerned in the production of the whisper, probably being held open during that process. If the main bronchus on one side be compressed or occluded, the voice-sounds are annulled on that side, and vocal vibrations, which are merely palpable sounds, are similarly obscured. If on the other hand the termination of the tubes be surrounded by a better conducting material, as in pneumonia, or if the tissue normally surrounding them be condensed upon them, as in that portion of a lung near the surface immediately above an effusion, the voice-sounds will become loud and sharply defined. Vocal resonance may be and is, no doubt, reinforced by a certain amount of reverberation or echo produced within the tubes, adding to its loudness but taking from it clearness and definition, for the conditions of true consonance can hardly obtain in an apparatus of which conduction of sound is in no sense a function. If a large bronchus, instead of being subdivided into innumerable smaller and diminishing branches, abruptly terminate in an empty cavity occupying the area of its former distribution, it is obvious that the voice and whisper sounds transmitted down it will be loudly heard over the cavity.

Ægophony is a voice-sound concerning the mechanism of which there has been much dispute. I have myself nothing to add respecting it, except that the sound appears to be the bronchophonic sound conducted through a thin layer of fluid. One may here notice a peculiar sign, to which attention has been drawn by Dr. Bacelli of Rome, whose observations have been noticed and confirmed by Dr. Gueneau de Mussy, now of Paris. The sound, *pectoriloquie aphonique*, is described as heard over the base of the chest in patients with pleuritic effusion, provided the effusion be of an homogeneous serous kind. On placing the ear over the back of the affected side, and making the patient repeat in a whisper some rough words, as "trente-trois," or "one," "two," "three," the sound is conducted through the fluid and up the stethoscope to the ear with great distinctness, the articulation being perfectly recognized. In cases of purulent effusions, on the other hand, no such conduction is to be obtained. Dr. Bacelli and M. Gueneau de Mussy regard this sign therefore as an important aid in distinguishing between cases of serous and purulent effusion. In my own experience it has sometimes proved useful and sometimes failed. Although a sign of a certain value it is not to be implicitly relied on.

Amphoric Resonance or metallic echo is a sound which is often heard in cases of pneumo-thorax. It is probably to be regarded as a reflected sound due to the impingement of vibrations against the walls of a large echoing cavity. The voice-sounds are not conducted through a pleura distended with air much more readily than through fluid, but a lung cavity in relation with such an air sac will transmit vocal vibration to it which may produce an echo. It is in cases in which the communication with the pleura is very small that this sound is best heard.

The cough sounds and laryngeal sounds heard in disease are of such obvious mechanism as to require no special comment.

THE MICROSCOPICAL EXAMINATION OF THE SPUTUM.

Valuable information may sometimes be obtained from a careful examination of the sputum, and in doubtful cases this aid to diagnosis ought not to be neglected; at the present time, when the intimate relation of micro-organisms to disease is a subject of such pressing interest, the help of the microscope becomes more and more imperative.

In examining sputum it is only necessary to spread a small portion on a glass slide and cover it with a thin cover-glass.

If the specimen be simply mucus, such as is secreted in health, a thin film will be observed, entangling air-bubbles, leucocytes, and occasionally a few ciliated epithelial cells derived from the air passages. By running a drop of acetic acid under the cover glass the nuclei of the cells become more conspicuous.

Beginners are very apt to mistake particles of dust, cotton fibres, pieces of feathers and foreign bodies of various kinds, which accidentally contaminate the fluid, for objects of graver import.

In bronchitis the sputum, which in the early stages is very frothy, becomes of a yellowish tint as the disease progresses. This change is due to the presence of a greater number of leucocytes, granular matter and oil globules. Blood corpuscles may occasionally be observed. If the disease be the result of irritant particles, as soot, dust, etc., pigment particles may be detected.

The sputum of pneumonia is characterized under the microscope by the presence of blood corpuscles uniformly diffused throughout the mucus.

In phthisis, when the lung is actually breaking down, shreds of pulmonary tissue may by careful manipulation be detected in the expectoration. For this purpose the expectoration should be collected for twenty-four hours, then boiled with an equal quantity of solution of caustic soda, (20 grains to the 1 oz.) and frequently stirred; the boiling fluid should then be poured into a conical glass vessel and freely diluted with distilled water. A more or less copious sediment deposits, numerous specimens from which should be diligently searched with the microscope for the boldly curved loops and fragments of pulmonary elastic tissue. (See Plate I, fig. 1).

Micro-organisms.—For the satisfactory detection of these minute bodies it is necessary to resort to special methods in the preparation of the sputum, and to employ powers magnifying at least 300 times.

The examination may be conducted on unstained specimens, but it is far more reliable to stain the specimen, which may be readily done by means of the aniline dyes sold for this purpose.

A method known as the Weigert-Koch method consists in spreading out a thin film of sputum on a slide or cover-glass, and drying by passing it rapidly several times through the flame of a spirit lamp. Several drops of the stain[1] are then poured on the specimen and allowed to remain for about fifteen minutes or more. The stain is then washed off with distilled water and afterwards with alcohol, and allowed to dry. It may then be mounted in Canada balsam. For staining tubercle-bacilli Dr. Heneage Gibbes's method gives very good results. (Full directions for preparing the stain will be found in the *Lancet*, May 5th, 1883, but it may be obtained ready for use from Messrs. R. and J. Beck, 68 Cornhill,

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[1] A ½ or one per cent. solution of either Gentian violet or Spiller's purple, in water, is recommended by Dr. Gibbes. Practical Histology and Pathology, 2nd edit., p. 135.

E.C.) It is used as follows:—The sputum in a very thin layer is dried on a cover-glass; a few drops of stain are next to be gently warmed in a test-tube until the steam rises and then poured into a watch-glass, and the cover-glass with the sputum placed upon the stain and allowed to remain four or five minutes. The specimen is then well washed in methylated spirit until no more stain comes away, then dried and mounted in Canada balsam as usual. If time be not important it is better still to dispense with warming the stain, and to allow the cover-glass to remain in the cold fluid for thirty minutes or longer.

The peculiarities of the various bacilli will be found described under the diseases to which they particularly refer.

Besides pus cells, leucocytes, blood corpuscles, and foreign bodies of various kinds, pieces of morbid growth, hydatids, echinococcus hooklets, and even mould, as *penicillium,* may be coughed up. Mould when present usually comes from the interior of phthisical cavities.

CHAPTER III.

DISEASES AND DEFORMITIES OF THE CHEST WALLS.

THE diseases of the chest-walls are of importance chiefly from the frequency with which they simulate more deeply seated lesions.

Pleurodynia, *πλευρὰ* the side, *ὀδῦνη* pain, may be due to *rheumatism*, *myalgia* or *intercostal neuralgia*.

The symptoms are very similar in all these conditions, but more particularly in the two first named, and there can be no doubt that aponeurotic rheumatism and myalgia are conditions frequently associated and difficult to separate. Pleurodynia commonly begins quite suddenly with severe pain of a tingling or burning character, much aggravated by respiratory movements, especially those of deep inspiration or tussive expiration.

The pain may be situated at any part of the chest. A common seat is over the centre, at and about the insertion of the pectoralis muscle on either or both sides, or at the margin or sides of the chest where the recti and serrati muscles are inserted. When the pain is lateral it is almost always limited to one side. The breathing of the patient is hampered and may even be greatly distressed by the severity of the pain.

Aponeurotic rheumatism is not common before thirty, although it may occur in young people of distinctly rheumatic diathesis. It is frequently attributable directly to exposure to cold, *e.g.*, coming out from heated rooms and walking or driving in evening-dress. Middle-aged and elderly people, who are the subjects of gout and allied rheumatic affections, lumbago, sciatica, etc., are specially liable to this affection.

Myalgia is, except so far as muscular tenderness is inevitably more or less associated with the preceding condition, generally traceable to some strain. It sometimes occurs in whooping cough and very commonly in the course of phthisis. Unquestionably the disease has in most instances a definite pathology in the rupture of some minute muscular fibres.

Intercostal neuralgia is a more definite ailment than either of the above, and occurs more especially at two periods of life, and in two forms. 1. It is very common in young females in the form of infra-mammary neuralgia, in which the pain is referred to the surface of the chest below the left breast. This affection is most generally associated with anæmia, attended with leucorrhœa, amenorrhœa, or other form of menstrual disturbance. The disease is very rarely seen in the male sex. 2. Intercostal neuralgia occurs also on either side of the chest in persons of middle or advanced middle life, and is more common in females. The pain is of a very severe stinging or burning character, along the course of the peripheral distribution of one or more of the intercostal nerves. It may last for days or even weeks before the appearance of the very characteristic lesion, *herpes zoster*, after the appearance of which the pain is soon mitigated or subsides altogether. This disease appears also to be commonly associated with the rheumatic or gouty diathesis. Although most common in middle-aged adult females, it is by no means limited to them, and may sometimes be observed in quite young people. The pain attendant upon *herpes zoster* in the young, however, is rarely severe, and I believe never precedes the appearance of the eruption by any considerable time.

The physical signs of pleurodynia, when carefully considered, are quite sufficient for diagnosis:—

1. The disease *per se* does not raise the temperature, in which fact we have one very important element in the diagnosis from pleurisy.

2. There is usually decided tenderness on pressure over the seat of pain. In intercostal neuralgia this tenderness is very superficial, and is best elicited by gently pinching up the skin; the tenderness may be observed, too, not only over the point most complained of, but also in the lateral spinal region corresponding with the distribution of the posterior cutaneous branches of the affected nerves. In these cases the situation is almost always below the left breast, and the patient presents the other physical signs of anæmia. In myalgia the pain is markedly elicited by bringing the muscles affected into action, *e.g.*, causing the patient to grasp the back of a chair, and to bring the pectoral muscles into action, or to raise himself in bed or turn on one side so as to involve other muscles. In aponeurotic rheumatism and in myalgia there is also tenderness on firm pressure over the muscles in the intercostal spaces, etc.

3. The respiratory movements are restrained by the pain, and respiration on the affected side is correspondingly weak, but it is of vesicular character and unattended with any rubbing sounds; one may sometimes hear, however, muscular rumbling sounds due to the voluntary restraint exercised upon this side by the patient. The percussion resonance is unaltered.

Pleurisy and pericarditis are the two conditions most simulated by pleurodynia. In phthisical subjects, where there is perhaps already existing pyrexia, it is often difficult to be sure that there is not some dry pleurisy, and here the diagnosis is not very important. Aponeurotic rheumatism of the front of the chest is distinguished from pericarditis by the absence of the physical signs proper to the latter complaint.

Treatment.—Intercostal neuralgia in young anæmic subjects requires general treatment by iron, quinine, arsenic, and fresh air, with generous diet; and local treatment, by soothing applications such as belladonna plasters, or chloroform liniment three parts with belladonna liniment one part, sprinkled on lint or flannel backed by oiled silk. Belladonna or aconite may also be efficaciously applied in the form of the solid liniments suggested by Dr. Sansom and prepared by Messrs. Savory and Moore. Menthol alone, or in combination with chloral hydrate or croton chloral hydrate, as suggested by Mr. W. Martindale,[1] is a valuable application in the *herpes zoster* form of intercostal neuralgia, before the eruption has appeared; afterwards the local remedies must be used with caution. In this latter form of neuralgia, however, the pain is often very severe and intractable, and sub-cutaneous morphia and atropine medication may be necessary. Local depletion by leeches will always give temporary relief, but in the anæmic form of neuralgia at least, it is inadmissible. The myalgic forms of pleurodynia are best treated by keeping the affected side of the chest at rest by strapping, as for dry pleurisy. Aponeurotic rheumatism may often at once be cured by the application of a well-made mustard poultice kept on for twenty minutes. In the case of any rheumatic diathesis, general remedies, especially iodide of potassium with quinine, will be required.

Perichondritis or *periostitis of the rib* is a disease sometimes giving rise to some perplexity in diagnosis. It is commonly due to syphilis, when the other manifestations of that disease may be observed. Direct injury is another frequent cause of this malady. A short time ago I had for some months under my observation, a lady in whom a thickening of the covering of the first two ribs in the left infra-clavicular region, was traceable to an attack of typhoid fever four or five months previously; local prominence, obscured breath-sound, some venous enlargement, and great tenderness were to be noted. In this case the thickening probably involved the fascial tissues between the ribs also, and there was much hesitation in diagnosis from local abscess or empyema, not only on my own part, but also by two very able surgeons, to whom I submitted the case. The lady ultimately, but very slowly, got well, under repeated and prolonged change of air and other remedies.

In some instances the first piece of the sternum is involved in periostitis, and when the periosteal thickening is intra-thoracic, as well as on the external surface, the symptoms and signs of thoracic tumor may be developed.

Amongst the thoracic parietal conditions that should be here noted is the very remarkable *fascial creaking*, that may not very uncommonly be heard especially over the supra-spinous and scapular regions. This phenomenon is attended with a certain amount of pain. I have met with more than one case of this kind which has been treated by very able practitioners, with great perseverance and activity by blisters and iodine counter-irritants for many weeks, under the impression that the case was one of chronic pleurisy. The condition is, however, so far as I have been able to observe, a permanent one, and is only important in diagnosis. If

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[1] Extra Pharmacopœia.

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the attention be once awakened to the possibility of the sounds being extra-thoracic, the diagnosis can be confirmed by making the patient stop breathing and by listening whilst the shoulder is shifted about.

Amongst the *deformities* of the chest, I would mention a deficiency of the clavicular portion of the pectoralis muscle, on one side of which I have seen two examples. A remarkable flattening or rather hollowing below the clavicle is thus occasioned, suggestive of grave internal lesion, the true cause of which is, however, at once rendered obvious by making the patient grasp the back of a chair with both hands and attempt to lift it, when the existing muscular fibres start into action and the defect is declared.

The shape of the chest is nicely conformed as a rule to the condition of the lungs. Thus we have the small long thorax, with oblique approximated ribs, associated with small lungs, the large expanded barrel-shaped chest with emphysematous lungs, the local flattening or enlargement with pulmonary or pleuritic diseases. Such conditions require no comment here. Nor, again, need we do more than make reference to the prominent upper chest of dorsal caries, the distortions corresponding to lateral curvatures of the spine, or the results of occupational pressure, *e.g.*, the hollowed lower sternum of shoemakers.

An area of the chest surface in the infra-mammary region on each side, having the fifth space in the nipple line for its centre, is unsupported by muscles, being an interval between the insertion of the pectoralis, the serratus and the rectus muscles. Thus in any obstruction to the entry of air into the lungs telling upon their general surface the atmospheric pressure is least supported by muscular action at these portions. Thus in young children, in whom the ribs and cartilages readily yield in any condition of general dyspnœa, as from croup, bronchitis, etc., this part of the chest on each side becomes depressed and the sternum tilted outwards. Repeated bronchial attacks thus give rise to the prominent sternum and depressed inferior and lateral thorax which persist and constitute the pigeon-breast.

In the *rickety thorax* a more or less deep groove corresponds on each side with the junction of the ribs and their cartilages, there being here a knuckling in, so to speak, of the ribs, causing the sternum to come forward with undue prominence.

The *alar thorax* of phthisis is produced by the small narrow thorax affording but insufficient breadth of support for the scapula, the superior extremities of which are tilted forwards and inwards by the weight of the arms and the inferior angle of the scapula thrown outwards, the effect on a thin subject being to produce a winged appearance when viewed from behind.

CHAPTER IV.

DISEASES OF THE PLEURA.

THE pleura on each side of the chest is a closed serous cavity or sac intimately applied to, and in organic union by its outer surface with the lung and the costal parietes. The internal surface of the sac is lined with endothelium. The costal and parietal portions of the internal surface are in close contact, lubricated merely by some moist serous secretion. This contact or apposition is maintained by atmospheric pressure bearing upon the interior of the lung and the exterior of the chest-wall, which more than counter-balances the constant tendency for the two surfaces of the sac to spring apart from the opposite elastic tractions of the lung and thoracic wall. In the cellular tissue subjacent to the pleura is a layer of lymphatics, which in the visceral portion freely inosculates with the pulmonary lymphatics, and communicates by open mouths (stomata) with the pleural sac. These lymphatics run their course towards the root of the lung.

When we regard the position and connections of the pleura, its extensive endothelial surface, the conditions of negative pressure in which that extended surface is ever maintained, its richness in lymphatics (indeed the pleural have, with other sacs of the kind, been regarded as lymphatic spaces), we cannot wonder that it should frequently become the seat of disease, nor that it should be one of the chosen sites for the manifestation of lesions from blood contamination. Few are the autopsies after adult age in which one fails to find some imperfections in the pleura—few are the cases of septic or pyæmic poisoning in which the membrane is not actively involved.

The diseases of the pleura are:—

(*a*) *Inflammatory.*—(1) Primary or simple (general or local), (2) secondary and septic, (3) suppurative.

(*b*) *Effusive.* — Hydro-thorax, pyo-thorax, pneumo-thorax, hæmo-thorax.

(*c*) *Growths and thickenings.*—Fibrous, tubercular, malignant.

1. SIMPLE PLEURISY.—Amongst the common causes of simple pleurisy, exposure to cold is the most frequent. The depression of temperature may be general, in persons insufficiently clothed, or whilst vital resistance is lessened from mental shock or insufficient food. Or the patient may receive a chill from exposure to draught or sudden change of temperature whilst over-heated. Certain diathetic states favor the occur-

rence of such pleurisies, *e.g.*, the rheumatic diathesis or albuminuria. But prior to the attack the patient may have been in good health.

Pathology.—The pathology of the disease may be briefly summed up as consisting in the first place of hyperæmia of the pleura, its vessels becoming minutely injected; in a very few hours the normal glistening appearance of the pleural surface is lost, it becomes cloudy as though breathed upon, and gradually covered with a layer of lymph. Both the costal and visceral layers are thus affected, and the effect of the movement of the two surfaces, covered with sticky lymph, upon one another is to roughen them, causing the effused lymph to present numerous little elevations and pittings. New vessels rapidly extend into the lymph from the pleural vessels, forming loops which, unless the opposed surfaces be speedily separated by effusion, meet and inosculate, and thus organic union takes place between them. But most commonly in simple pleurisy an effusion of liquor sanguinis takes place from the vessels, and collects in the pleura, thus separating, more or less widely from below upwards, the pleural surfaces. In due time, and in ordinary cases, the acuteness of the disease passes away, the fluid effusion is again absorbed, the lymph-covered surfaces come again in contact and rapidly adhere, the originally effused lymph becoming in course of time degenerated and absorbed.

Symptomatology.—The symptoms and signs of pleurisy are in accordance with its pathology, and are divided into three stages, viz:—1. The stage of hyperæmia and commencing exudation. 2. The stage of effusion. 3. The period of absorption and convalescence.

Pain in the side and rigors are the two symptoms which usher in an attack of pleurisy, and either may precede the other by a few hours. The pain is that of an acute "stitch" in the side, usually felt in the lower axillary or infra-mammary region, but not uncommonly referred, and especially so with children, to a much lower point in the abdominal wall, to which the terminal cutaneous twigs of the affected intercostal nerves are distributed. The pain interferes with the respiratory movements, which are restrained and shallow, the patient inclining to the affected side so as to lessen its movement. The rigors are of variable severity, sometimes very sharp and decided, at other times amounting only to recurring chills. It is stated that the rigors of pleurisy are repeated, whilst in pneumonia one severe shivering occurs at the commencement of the disease: on neither hand, however, is this statement more than generally speaking correct. There is occasional dry interrupted cough. The temperature is elevated usually to about 102° or 103°, the face anxious, pale, the pulse small and moderately frequent. The fever has never the marked character of that of pneumonia, and the flushed cheek so characteristic of the latter disease is rarely present. In a word the patient with acute pleurisy is not so ill as one with pneumonia, although he may be in more suffering.

In the first stage of the disease there is no percussion dullness. On listening to the chest the respiratory murmur will be found to be uneven and partially suppressed on the affected side, and more or less distinct friction sound, usually of a grating character, is heard most distinctly at the end of inspiration and the beginning of expiration, over the seat of pain, usually in the mammary or infra-mammary region or over the base of the lung. In left-sided cases friction, of cardiac rhythm, may be heard over the precordial region. Friction-fremitus is occasionally to be felt. In some cases which present all the symptoms and febrile phenomena characteristic of pleurisy no friction sound can be heard; this may be explained by the manner in which the affected side of the chest is held fixed by the patient, or by the pleuritic exudation commencing on the diaphragmatic or mediastinal aspect of the pleura, and thus being beyond the reach of appreciation by the stethoscope.

In a certain number of instances the disease stops short at the dry or plastic stage, and the patient is said to have had a dry pleurisy; this however, though common enough in secondary and especially in tubercular pleurisies, is rarely observed in cases which have presented in any marked degree the features of acute illness characteristic of an attack of simple pleurisy.

Within a short time, it may be but a few hours, fluid commences to be effused, and dullness may be detected at the extreme base posteriorly, gradually extending upwards towards the apex. With the occurrence of effusion the pain becomes less, the breathing easier and less catching, although quicker than natural. The movements of the affected side are notably lessened, whilst in marked contrast to the effacement of respiratory sounds on the affected side is their exaggerated intensity on the sound side. In moderate degrees of effusion the dullness varies slightly with the position of the patient; whilst lying down, for instance, the resonance may be good to just below the nipple, whereas on sitting up there is dullness up to this point, and it may be higher. Over the lower portion of the dull area the respiratory murmur is absent, and the friction is no longer to be detected; but as the upper limits of dullness are approached in the scapular region, distant tubular breathing may be heard, and friction sound of a moister character is audible, especially in front.

As the effusion advances the breathing becomes increasingly distressed until apnœa is threatened.

The signs of pleuritic effusion may be divided into three groups.—(*a*) The cardinal signs of pleuritic effusion, the presence of which is alone essential for diagnosis, viz:—(1) Percussion dullness. (2) Displacement of the heart. (3) Annulled vocal fremitus. (4) Diminished and altered or absent breath-sound. These signs are common to both serous and purulent effusions.

(*b*) Subordinate or supplementary signs, viz:—(1) Increased semi-circumference of chest. (2) Intercostal bulging, elasticity or fluctuation. (3) Skodaic resonance. (4) Altered voice-sound. (5) Displacement of abdominal viscera. (6) Signs in the other lung. (7) Cardiac displacement bruits. These signs are none of them essential for diagnosis; any or all of them may be wanting.

(*c*) Signs indicative of nature of fluid, viz:—(1) Pectoriloquie aphonique (Bacelli). (2) Temperature signs. (3) Other pyrexial or septic phenomena. These are of especial importance with regard to the diagnosis of empyema, and will be discussed under suppurative pleurisy.

1. The *dullness* of effusion is absolute and toneless. It is distinguished by our American *confrères*[1] from more ordinary degrees of dullness by the term "flatness." It is the dullness of a brick wall rather than that of a table. In ascertaining the limits of dullness in cases of effusion, very light percussion should be employed, or the resonance of neighboring parts will be elicited.

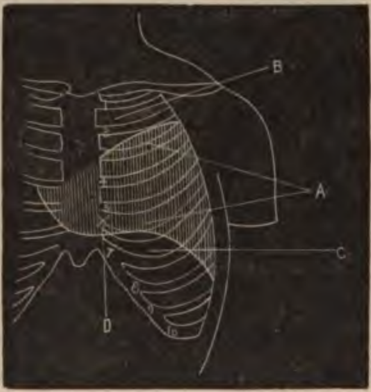

FIG. 6.—Percussion signs in case of moderate effusion. A, area of complete dullness ("flatness"); B, area of tympanitic (Skodaic) resonance; C, inferior curved line of tympanitic (stomach) resonance.

In cases of moderate effusion, the lung being texturally healthy, the upper margin of dullness in front is not a level line, but slants downwards and inwards in such a manner as to leave a somewhat triangular space of resonance (B, Fig. 6), the apex of the triangle being at the sterno-clavicular articulation. The resonance within this area is of a peculiar tympanitic quality, termed Skodaic resonance, to which I will again refer. The line of dullness (flatness) is highest in the axilla, and extends round the scapular region until in the interscapular region it again slants downwards, a tongue of comparative resonance (B, Fig. 7) protruding downwards in the postero-median line, which has been described by Dr. Garland as the "dull triangle,"[2] in contrast with the flatness or absolute dullness of the percussion-note external to it. Even in cases in which the effusion is very considerable and of old standing, the triangular area of comparative resonance (but now somewhat tubular in quality) can still be made out at the sterno-clavicular angle, and also in the upper interscapular region. The curved line marking the upper limit of dullness posteriorly, is sometimes spoken of as the letter S curve of Dr. Ellis,[1] and Dr. Garland maintains that the curve invariably commences at a lower level behind than in front. I have not found this to hold good, however, in all cases. The line of dullness does not very notably shift (although in this respect cases vary within narrow limits) with the position of the patient. In cases of extreme effusion, the "flatness" of the percussion-note extends throughout the utmost limits of the thorax, and, of course, in such cases these triangles of resonance are not to be found. In other cases, again, œdema or consolidation of the lung may obscure the signs over the areas referred to.

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[1] Austin Flint, "On Percussion and Auscultation."

[2] "Pneumono-Dynamics," by G. M. Garland, M.D., New York, 1878, page 15.

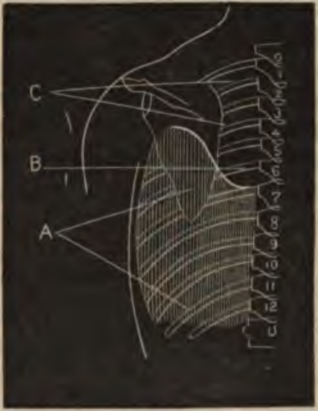

FIG. 7.—Percussion signs in case of moderate effusion. A, area of complete dullness ("flatness"); B, pulmonary note usually impaired; C, more or less tympanitic high-pitched resonance.

Thus the upper margin of the effusion, in typical cases, is not a water-level, but presents a curve, having its convexity upwards and in the lateral region, contrasting in this respect with the upper limit of dullness in cases of hydro-pneumo-thorax. As the fluid increases the dullness encroaches upon the median line, extending farthest across at the third and fourth cartilages, but being continued further outwards below this level by the outline of the displaced heart (D, Fig. 6).

The lower limit of dullness in typical cases of effusion is again peculiar (C, Fig. 6), the arch of the diaphragm being still preserved, even when the effusion is very considerable—so long, indeed, as Skodaic resonance is manifested. This can be best observed in left-sided cases, and to it I will again allude immediately.

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[1] Boston Medical and Surgical Journal, January 1st, 1874, p. 13, and December 14th, 1876, p. 689; quoted by Garland.

2. *Displacement of heart towards the sound side* is the second cardinal sign of pleuritic effusion. The absence of this sign would negative the diagnosis of unilateral effusion, unless it were explained by some special circumstances, *e.g.*, retention of the pericardium by old adhesions, the result of some former disease, or consolidation of the opposite lung. But these and other causes of fixity of heart are rarely met with; the mediastinal folds are ready to shift to one side or the other, instantly or slowly, in obedience to the rapidity or slowness with which the pleura on one side is occupied, and the lung on that side collapsed, leaving the traction of the other lung upon the mediastinum unopposed. I venture to lay much emphasis upon the displacement of the heart, rather than on displacement of other organs, as a sign of pleuritic effusion, because it takes place immediately and *pari passu* with the effusion. In this respect it strikingly differs from displacement of the diaphragm and abdominal viscera. The diaphragm is held up in the arched position until the effusion has mounted to a considerable height, and when the heart has already markedly retreated towards the opposite side. In fact, displacement of abdominal viscera is no essential sign of pleuritic effusion, and is only present in extreme cases; whereas displacement of heart *is* an essential sign, and (unless prevented by countervailing causes) is present from the first in all cases of unilateral effusion.

Although the exact position of the heart is often somewhat difficult to make out, the trouble taken is well rewarded by the importance of the sign. The position of the apex beat should be carefully felt for by the hand, and, if necessary, by the ear through the medium of the stethoscope. Percussion may also be usefully employed to trace from the sound side the line of cardiac dullness.[1] The axis of the heart is not greatly changed in direction by any common degree of effusion; it becomes a little more vertical, and in very extreme cases it may become slightly twisted. I have never seen, nor have I been able to produce by experiment, anything approaching to the complete turnover of the heart represented in some books as occurring in effusion.[2] In cases in which the heart is greatly displaced, a systolic murmur is developed over its base, which disappears on removal of a portion of the fluid, and is presumably due to straightening or slight tension of the great vessels from pressure of the fluid.

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[1] There is one fallacy with reference to cardiac displacement in the earlier stages of effusion that may be here noted—viz., that as the base of the lung retracts, the right or left margin of the heart (as the case may be right or left-sided) becomes uncovered: this may lead to an apparent delay in the displacement of the organ, the more extreme left or right boundary thus being within reach of palpation.

[2] Notes on Displacements of the Heart. By the Author. British Medical Journal, July 17th, 1869.

3. *Absence of vocal fremitus* is a third very important sign of pleuritic effusion; it is obviously due to the intervention of a bad conductor of such vibrations between the vibrating media and the chest walls. If these two latter be united at any point by a cord of adhesion, the fremitus may there be felt. Just as light percussion is needed to define the exact limits of dullness, so light palpation is necessary, employing the finger-tips rather than the whole hand, in order better to exclude vibrations from above as the confines of the effusion are approached.

It will thus be observed that the three most essential signs of pleuritic effusion are elicited by palpation and percussion.

4. The stethoscopic signs in pleuritic effusion are sometimes misleading. A certain value is justly attached to the *absence of breath-sounds*, and (except towards the upper confines of the effusion) of voice-sounds in the diagnosis of pleuritic effusion; but in some of the most typical cases of this disease the breath-sounds are audible over the whole side, well defined, tubular, and not easily distinguished from the breath-sounds of pneumonia. The voice-sounds may also be well conducted over the whole side, and the whisper exaggerated even to pectoriloquy. In September, 1881, a boy, aged eleven years, was admitted into my ward at Middlesex Hospital, with acute pleurisy and effusion on the right side of four days' duration. On the fifth day after admission, the dullness had extended to the clavicle and well over the median line. The vocal fremitus was annulled, the heart was beating considerably outside the left nipple line, and the cyrtometer tracing showed enlargement of affected side, but *tubular breath-sound and whispering pectoriloquy* could be well heard posteriorly to the base, and somewhat more feebly over the lower two-thirds anteriorly. It was difficult to believe that one was listening over an immovably compressed lung, separated from the ear by some inches of fluid. Twenty-six ounces of clear serum were, however, readily removed the next day, with great relief to the little patient. This is no isolated case, and I am inclined to believe that the not infrequent presence of these auscultatory phenomena leads to the somewhat common diagnosis of "pleuro-pneumonia," a combination—I mean in the sense now employed of pneumonia with effusive pleurisy—in my experience of rare occurrence. The reasons why we should have absence of breath-sound in pleuritic effusion seem so ready to hand, that an exaggerated importance has perhaps on this account been attached to this negative sign—for (1) a certain thickness of fluid separating the lung from the parietes, and (2) the lung being collapsed and immovable, how should it yield any respiratory sounds? Although the first explanation is a valid reason why the respiratory sounds should be weakened and distant, yet it must be remembered that fluid is by no means a bad conductor of sound, and particularly the

more homogeneous kinds of fluid.[1] The second suggestion well illustrates the falseness of the still prevalent views as to the production of breath-sounds. It is impossible, with a due regard to clinical observations of such cases, to hold that the breath-sounds (with the single exception of the *vesicular portion* of the normal respiratory murmur) are generated within the lungs or bronchi. They are really laryngeal (glottic) sounds conducted down the tubes and through a certain thickness of residual (tidal) air. Over the sound side in the boy just referred to, the laryngeal breath-sound was heard together with the vesicular sound as the soft (but exaggerated) respiratory murmur, but over the effusion the tubular sound was alone heard, imperfectly conducted through solid and fluid media.

Supplementary signs of effusion.—Much importance has always been attached to increased semi-circumference as a sign of effusion. On inspection there is generally an apparent enlargement of the affected side, whilst to actual measurement there may be little or no difference. Any relative enlargement that may be present is more obvious during deep expiration. The *total* circumference of the chest is, however, always increased in effusion. In any case in which we obtain any decidedly increased measurement on the affected side, we may be sure that the effusion is a large one. The *shape* of the affected side is altered in effusion, being more rounded. This is well shown by a cyrtometer tracing, and arises from the fact that the outspring of the ribs is entirely relaxed on the affected side (even if there be no positive intra-thoracic pressure), whilst on the sound side it is only partially so. When we bear in mind that the capacity of a globe is greater than that of any other shaped figure, and further remember that, in pleuritic effusion, the heart is displaced to the sound side, it is clear that in all cases of effusion, and quite apart from any relative increase in circumferential measurement, there is increased capacity on the affected side of the thorax.

Cyrtometer tracings thus give us more information than tape measurements, since they also take into account altered shape.

Intercostal dullness or *fluctuation* is a sign of inconstant and rather exceptional occurrence in cases of effusion, intra-thoracic pressure alone being insufficient to cause it. It is more commonly present in children than adults, and especially in weakly children with wasted muscles. Ataxy of intercostal muscles, and a certain degree of laxity or softening of thoracic pleura, are needed for the presence of this sign, which, in my experience, has been more often associated with purulent than with simple effusion.[1]

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[1] I have had recently at Brompton Hospital a case of pleuritic effusion on the left side, in which an endocardial mitral murmur was well heard through the fluid below the angle of the scapula—as well conducted as it would be through lung. It is a question whether the complete absence of breath-sound in some cases of pleuritic effusion is not attributable to partial or complete occlusion of the bronchus by accumulation of mucus.

Ægophony is a sign commonly met with in pleuritic effusions, and is of some value in the diagnosis of localized effusions from consolidation. It will often be observed on listening for ægophony in cases of acute effusion that a certain lisp attends the voice-sound, which lisp is more sharply conducted than the ægophonic sound. In such cases, if the whispered voice be listened to, it will be found to be very distinctly heard through the fluid. (*Vide* p. 64, Bacelli's sign).

Pressure signs.—The presence or absence of *pressure signs* in pleuritic effusions is a matter of importance in determining whether or not interference by removal of the fluid is called for. It has been already pointed out (page 82) that whereas cardiac displacement is an essential sign of effusion, it is no evidence of intra-thoracic pressure. Displacement downwards of the diaphragm is on the other hand always a sign of intra-pleural pressure. The relationship of the diaphragm to the cavities above and below it, is quite a different one to that which the mediastinum bears to the pleura on either side. We have on the thoracic side of the diaphragm, under normal conditions, a negative pressure; on the abdominal side the prevailing pressure is positive, although there may at times possibly be a negative pressure, which can never, under any circumstances, contend against the elastic traction of the lungs.[2] The negative pressure is maintained within the pleura until the lung has completely contracted before the advancing fluid; and after this point any further accumulation compresses the lung, and by its weight and pressure forces down the diaphragm. Hence displacement downwards of the abdominal viscera is a late phenomenon in pleuritic effusion. It also varies according as the patient has been keeping about or in bed. Further, it is more often met with in purulent than in serous cases; and possibly this may (if it be the fact, as my observations would lead me to think) be explained by the nutrition and elasticity of the lung becoming damaged by the more severe fever attendant upon suppuration. It is obvious that mere loss of tone in the diaphragm would not, as in the case of the intercostals, account for the difference. I have found the intra-thoracic pressure to vary from a negative pressure to ½ in. and 1½ in. positive pressure of mercury at the commencement, and at the termination of paracentesis from ⅛ in. to ½ in. and even 1 in. negative pressure, there being in all cases a more or less considerable amount of fluid still remaining in the pleura.

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[1] Dr. Stokes draws attention to the fact that intra-thoracic pressure alone is insufficient to cause bulging of the intercostal spaces, which he considers as rather due to inflammatory paralysis, and as significant of severity of lesion, and as therefore of value in prognosis. Vide Stokes, "On Diseases of the Chest," New Sydenham Society's edition, 1882, pp. 485, 512.

[2] The outspring of the lower ribs tends, through the diaphragm, to act as a counterpoise to the upward traction of the lungs.

The clinical outcome of these observations upon the displacement of the diaphragm is of very great practical importance—viz., that, as was first pointed out by Traube, and more fully explained in an admirable monograph by Garland of Boston, stomach-note may be obtained at the sixth rib in the nipple line in the presence of a large effusion on that side. Similarly, on the opposite side, under the same conditions, the liver-dullness may not be appreciably lowered. These facts are to be borne in mind in choosing the spot for paracentesis, otherwise awkward accidents may happen.

My observations would lead me to assert that, in recent cases, the period of effusion at which the intra-thoracic pressure is converted from a negative pressure or zero to a positive pressure upon the lung and heart is marked clinically (1) by the dullness mounting up above the third cartilage (patient in sitting posture), and (2) by the Skodaic resonance becoming changed from the full note to a more tubular quality, and finally becoming extinguished.

Skodaic resonance is thus of some clinical value in guiding us to a judgment as to the presence or absence of pressure within the thorax, and the advisability or urgency of interference by paracentesis. Different views have been held respecting the mechanism of Skodaic resonance in pleuritic effusion. Skoda himself regarded it as the relaxed lung note, due to the lung contracting upward towards its root as the fluid advances, and, when in contact with the parietes, yielding a note on percussion similar to that given out by a healthy lung on the post-mortem table. My colleague, Dr. R. Thompson,[1] regards the note as significant of a lung still in contact with the parietes at the upper part of the chest, but slightly on the stretch, its periphery being adapted to the somewhat wider arc assumed by the ribs when released from the lung traction below. Whichever of these explanations we adopt (and they are probably both accurate at different stages of the effusion), it is obvious that a slightly negative or at least a zero pressure is essential for the Skodaic resonance of this mechanism — the full-toned Skoda note. On the other hand, we often meet with cases of effusion in which at, and near, the sterno-clavicular angle a tubular quality of resonance is obtained, resembling the percussion note over a considerable lung-cavity, or over the cheek held on the stretch with the mouth slightly open. This resonance corresponds, I believe, with that explained by Hudson[2] as produced by the lung being collapsed upon its root, and thus yielding the tracheo-bronchial resonance. This note clinically signifies a higher degree of effusion than the other; it means that the lung is not simply contracted, but *collapsed* by the *pressure of the advancing fluid.* In the next degree of effusion all resonance of whatever kind is everywhere lost.

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[1] "Physical Examination of the Chest," p. 49.

[2] Quoted by Walshe, "Diseases of the Lungs," fourth edition, p. 74.

In addition to the *displacement murmur* over the heart already alluded to, another sign of intra-pleural pressure, of more importance, is the occurrence of crepitant sounds over variable portions of the sound lung. These congestion râles give evidence of great stress of circulation in the sound lung, and are associated with a congestive cough, and a blood-streaked viscid sputum. With the development of such signs the dyspnœa becomes increasingly urgent, the surface dusky, and the patient is in imminent danger from syncope.

It is not usual, however, for the effusion to attain such dangerous proportions; in a large number of cases the area of Skodaic resonance is not encroached upon, when the tide turns and absorption of the fluid commences.

The fever of acute pleurisy subsides in from a few days to a week or a fortnight, according to the severity of the attack; the tongue cleans, the appetite returns, and the patient has only to gain strength and to await recovery from the results of the local inflammation and effusion. In favorable cases, as the fluid becomes absorbed, the lung expands from above downwards, the pleura friction-sound returns, and with it usually some pleuritic pain, although of not nearly so intense a character as in the first instance.

Dr. Gee, in his work on Auscultation, has very minutely and accurately described the physical signs and other phenomena attendant upon recedent effusions; suffice it here to say that the respiratory murmur slowly returns, at first in the upper portion of the chest, and the heart gradually resumes its normal position. The absorption of the last portion of fluid is often considerably delayed, and dullness and weak respiration may long persist at the posterior bases. In some cases the effusion may show no signs of becoming absorbed, in others again complications may arise. But in the great majority of instances the *prognosis*, in cases of simple acute pleurisy, is decidedly good.

The *diagnosis* of pleurisy is not attended with any great difficulty. As regards *pain* it may be simulated by pleurodynia, or intercostal myalgia, neither of which affections is, however, attended with febrile phenomena, nor with friction sounds nor the signs of effusion. Pneumonia is the disease with which pleurisy is more often confounded, and we must observe that the two may co-exist. The absence of rapidly supervening bronchial respiration with bronchophony and fine crepitation, the freedom from blood-stained sputa, and the gradual effacement of respiratory sounds and of vocal fremitus, with increasing displacement of heart as dullness on percussion increases in extent, render the diagnosis of pleurisy with effusion certain.

The chief difficulty in diagnosis is, however, to ascertain the probable *nature* of the pleurisy, whether simple with serous, or suppurative with purulent, effusion. I will postpone the discussion of this point until the consideration of the graver malady.

The *treatment* of acute pleurisy of ordinary severity does not call for any very energetic measures. In the first stage of the disease our object is to reduce arterial pressure within the vessels of the diseased pleura—vessels which are temporarily paretic and in a state of acute turgescence. One or two doses of aperients, and action upon the skin and kidneys by saline diaphoretics and diuretics, are useful general measures to this end. At the first onset of this disease small doses of aconite may sometimes be given with advantage, with the view of reducing arterial pressure, but with the commencement of effusion this remedy should be stopped. The pyrexia of pleurisy never of itself causes anxiety. Rest in bed must be absolutely enjoined, even in the least severe cases, and the diet restricted to nutritious fluids. There is no object to be gained by restraining the patient from taking bland drinks to ease thirst. The application of leeches to the side is often of great value in lessening the intensity of the local lesion as judged of by pain and pyrexia. Hot poultices, frequently changed, are also valuable, serving by the dilatation of superficial capillaries to ease pressure upon the deep distribution of the intercostal arteries. A blister applied under the poultice is sometimes useful. A fair amount of sleep must be produced by the judicious administration of opiates, which may be given in the form of Dover's powder, or *in adults,* if the pain be very severe, by subcutaneous injection. The temperature of the patient and the signs of effusion must be carefully watched.

Stage of effusion.—Whilst the inflammatory fever is at its height, the less we meddle with any effusion present—unless it become of itself a danger—the better. We must bear in mind that a certain amount of effusion is as much to be looked for in acute pleurisy as exudation into the air-vesicles in pneumonia, or "running at the nose" in nasal catarrh; and the products in the three cases do not, *cæteris paribus,* essentially differ. The pulmonary exudation consolidates *in situ;* the nasal product stiffens the handkerchief; and the exudation into the closed pleural sac remains limpid and liquid only until exposure to air or the presence of a foreign body (or perhaps intrinsic change from lapse of time) determines a certain variable amount of coagulation, a thin layer of which covers the roughened surface of the pleura and protects it, and flakes of which sometimes float in the fluid. Again, given acute inflammation of the coverings of the lungs, a certain amount of effusion is useful in separating and bathing in a bland fluid the tender and inflamed surfaces, and further, in keeping at rest the affected portion of lung. It is too often forgotten that the lung is in health exercising a constant traction upon the pleural sac, the vessels of which have therefore to sustain a negative or aspiratory pressure; this being so, it is but natural and physiological that, if these vessels become temporarily weakened and congested by the inflammatory process, increased exudation should proceed from them. The effect of this exudation is to neutralize lung traction, and therefore to lessen afflux of blood to the weakened vessels. It is, moreover, the surest and most natural means of giving that rest to the inflamed surfaces which they need for recovery. The effusion reaches its acme, the fever subsides, and in a few days the tide turns, re-absorption being effected in a few weeks.

Fluid effusion being thus both natural and salutary in acute pleurisy, we must be watchful, but not meddlesome, in our treatment of its earlier stages. Up to the end of a week or ten days we need not, in ordinary cases, consider how to promote its removal; and, in the majority of cases, after this period the fluid will gradually subside by spontaneous absorption. Counter-irritation is of undoubted value at this stage, and small doses of iodide of potassium may be added to the prescription. As the absorption is being effected, and when pain arises from the pleural surfaces again coming together, the firm application of a broad band of adhesive plaster round the affected side, or of several bands in the manner suggested by Dr. Roberts,[1] extending well beyond the median line in front and behind will relieve pain, and facilitate adhesion. Mineral acid tonics, usually with iron, are now required, and change of air will hasten the complete re-absorption of fluid, and the restoration of the function of the lung.

Towards the end of the second or third week of illness, if absorption be not in fair progress, the question arises whether it be advisable to interfere and remove a portion of the fluid by paracentesis. Thanks to the labors of Hamilton Roe, Trousseau, Bowditch, Dieulafoy and others, this operation has been rendered safe and easy of performance, so that we may consider the advisability of artificially removing the fluid from the chest, without being harassed in our judgment by the fear of so doing. The extent of the effusion, the condition and family history of the patient, and the nature of the fluid effused, are the points which determine for or against the operation, and the following propositions seem to me to be best justified by experience.

1. With good Skodaic resonance down to the third rib, and with no material enlargement of the side, we may assume, as I have already pointed out, that, although much fluid be present, the lung is only held in the position of physiological rest, and that therefore operative interference is not called for.

2. The continuance of pyrexia would, at the end of the second week, at all events, dispose us to wait longer. If no progress in absorption has been made, however, by the end of the third week, we may with propriety remove a portion with the aspirator.

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[1] Theory and Practice of Medicine, 5th edit., p. 453.

3. If the family history of the patient be unfavorable, our inclination would be to early interference, lest interstitial changes occur in the lung unfavorable to its re-expansion. [In cases, however, in which the effusion has occurred on the side on which there is already existing lung-disease of a phthisical nature, we should be *loath* to interfere, as experience certainly shows that an effusion checks, and sometimes arrests, the tubercular process.]

4. In cases in which the effusion mounts up to the second rib, or higher, extinguishing Skodaic resonance, and causing decidedly increased measurement of the side, we may be sure that there is positive intra-thoracic pressure, impeding the heart's action, and compressing the lung so as to retard circulation through it. In such a case the pressure may amount to one inch or one inch and a half of mercury. Under these circumstances the patient is in danger of syncope. The signs especially indicative of danger, viz: straining, retching cough, with frothy, viscid sputa, sometimes streaked with a speck or two of blood, and the presence on the healthy side of rather fine crepitant râle, have been already pointed out. In the presence of such signs, no matter what the stage of the disease, immediate interference is called for. The removal of two or three pints of fluid by the aspirator or syphon will give the patient immediate relief, and no further operation may be necessary. It has been well pointed out by Dr. B. Foster, that the symptoms of danger, lividity, palpitation, etc., are often in abeyance whilst the patient is perfectly still.

5. In cases in which there is any reason to suspect that the fluid may be purulent—quite apart from any question as to its amount—the needle of a fine subcutaneous syringe must be inserted at about the sixth space mid-axillary line, and a sample removed for inspection. Should it be pus of healthy character and sweet, steps must be taken for its thorough evacuation within a few days. Provided the pus be perfectly sweet, it is, I think, best not at once to operate upon a large effusion by making a free opening. The lung is compressed, and it is better in the first instance to remove a portion of the fluid by means of the syphon, for the double purpose of re-expanding the lung-texture, and diminishing the cavity of the abscess to be subsequently (within a short time, however) dealt with. Dr. Foster has also insisted upon the advantage of thus proceeding.[1]

LOCAL PLEURISY, PLEURISY FROM EXTENSION.—Although local pleurisy sometimes occurs as a primary disease from exposure to cold, it is much more commonly determined by neighboring disease; and conversely, pleurisy from extension is in most instances of localized and restricted area. Local pleurisy may arise from injury or disease of the chest-wall, *e.g.*, a blow on the chest, fractured rib, syphilitic periostitis, cancer of the breast, or from subjacent disease of the lung, hæmorrhagic infarct, gangrene, pneumonia, tubercle or cancer.

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[1] "On the Treatment of Pleurisy and Pleuritic Effusion." Address to the North Wales Branch of the British Medical Association, by G. Balthazar Foster, M.D., F.R.C.P.

The pathology of local pleurisy is, except for any associated lesions, the same as that of simple pleuritis, but it does not usually proceed to fluid effusion. As a rule the two opposed surfaces pass through the stages of injection, of lymph exudation and then either adhere and enter into organic union, or become smoothed down to present hereafter localized thickened opacities. A local pleurisy, however arising, may extend and involve the whole pleural sac. The course of local pleurisy is generally favorable.

Treatment.—In nine cases out of ten immediate relief is given by firmly strapping the affected side, so as to restrain movements, and in simple cases this may suffice for the cure. In some other cases, however, the pressure of strapping cannot be borne, either on account of its increasing the pain, or from the fact that the pleurisy has supervened on the side most available for respiration, as in certain cases of phthisis. Under such circumstances the application of a small blister, with or without a hot linseed poultice superposed, will give relief, or if the pain be very severe a couple of leeches may be applied. A very useful clinical test as to the amount of relief that may be expected from strapping in any given case, is obtained by observing the effect of steady and firm pressure upon the side by the hand. An opiate, or morphia subcutaneously, may be employed in association with local remedies, but it is much better practice to arrest a lesion, than merely to lessen pain from it.

2. SECONDARY PLEURISY, SEPTIC PLEURISY.—Many pleurisies which are spoken of as simple are really secondary to some morbid constitutional conditions, *e.g.*, the rheumatic diathesis; some pleurisies are so distinctly rheumatic as to require special treatment. Pleurisy, like other serous inflammations, frequently arises in association with albuminuria. These two pleurisies, the rheumatic and the albuminuric, are generally attended with copious effusions, sero-fibrinous, rarely purulent, rich in fibrin in the former, and in this variety the disease is also attended with profuse sweatings. It is not uncommon to get pleurisy, sero-fibrinous, as a complication in jaundice. The pleuræ are frequently involved in general outbreaks of miliary tuberculosis.

In septicæmia and pyæmia pleurisy is commonly present, and under these conditions the lymph and fluid exuded are highly charged with pus elements.

3. SUPPURATIVE PLEURISY.—Suppurative inflammation of the pleura is a far graver malady than any we have yet considered in this chapter. Whereas in acute simple pleuritis the inflammatory products consist of simple fibrinous exudation, which coagulates in a thin layer upon the

pleural surfaces, and of liquor sanguinis, which exudes through the weakened vessels and collects in the pleura; in suppurative pleurisy, on the other hand, we have a more intense and a less sthenic process, accompanied by the exudation of a more corpuscular and less coagulable lymph, and of liquor sanguinis rich in leucocytes. The one case—simple exudative pleurisy, is perfectly analogous with sthenic exudative pneumonia, the other—suppurative pleurisy, with the corresponding disease occasionally met with under greatly depressed conditions of system, whether from intemperance or other causes, viz., purulent infiltration of the lung; and just as in pneumonia, the simple may pass into the suppurative inflammation, so it is not unknown in pleurisy for a hydro-thorax to become an empyema. Fraentzel,[1] indeed, denies the occurrence, save on very rare occasions, of an effusion purulent from the first. "In almost every case the effusion is at first fibrino-serous" becoming subsequently purulent. Unquestionably serum is more easily effused than pus, and purulent effusions are at first thin and diluted, but the pus element is from the first largely present and active in acute empyemata.

What the conditions are that determine suppurative rather than simple pleurisy we do not precisely know, and we have to speak of "depressed conditions of the system," "morbid constitutional states," and the like; expressions which, nevertheless, are in keeping with our knowledge and are generally understood. We may, indeed, with some plausibility maintain that some septic agent present in the blood renders the inflammation purulent rather than serous, as in the joint affections in pyæmia, although the pus-producing quality in the blood is very difficult to estimate, and would seem to be of varied sorts. In such diseases as pyæmia, scarlatina, typhoid fever, and in the puerperal state, pleurisy, when it arises, is most generally suppurative; in rheumatism, gout, albuminuria, and delirium tremens, it is, on the other hand, as a rule, simply serous. Empyema is again very apt to occur in cases in which disease of the lung is also present. Perforation of the pleura in phthisis almost always leads to pyo-thorax, no doubt principally from the escape of purulent matter into the sac. Bacilli have been found in tubercular purulent effusions, and MM. Chauffard and Gombault[2] have found that the fluid of sero-fibrinous pleurisy in tubercular subjects when introduced into subcutaneous tissues of guinea pigs will, in a certain proportion of cases, lead to tuberculosis.

The *symptoms* of suppurative pleurisy do not very strikingly differ from those of serous effusion; in many cases from symptoms and signs alone it is impossible to make a certain diagnosis. The symptoms are commonly less acute but more adynamic in character than in ordinary pleurisy. The rigors or chills are more persistently recurrent, the daily fever continuing week after week; the pulse is from the first more frequent than in serous pleurisy, and keeps up its frequency throughout the disease. The tongue is more furred, with a greater tendency to become dry and brownish in the centre. The general condition of the patient is more depressed and anxious, and emaciation is much more rapid than in ordinary pleurisy. But perhaps the most important sign of the effusion being purulent (although not absolutely characteristic), is the occurrence of hectic sweats breaking out whenever the patient falls asleep.

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[1] Ziemssen's Cyclopædia, vol. iv., p. 611.

[2] Bull. et Méms. Soc. Med. des Hôp. de Paris, 3me série, I., 1884, p. 309.

The *physical signs* must necessarily be for the most part the same, whether the case be one of simple or suppurative pleurisy. Are there any physical signs, however, which are characteristic of pus in the pleura? Œdema of the chest-wall when it occurs, is, I believe, an absolute sign of the presence of pus. An erysipelatous blush over a portion of the chest, or still more evident pointing, are also sure signs of the effusion being purulent. But these are late signs, and therefore of greatly mitigated value. It is maintained by Dr. Bacelli of Rome,[1] that by the mode of transmission of vocal vibrations, the diagnosis between empyema and hydro-thorax may be made with certainty, whilst all other evidence of duration of disease, œdema, emaciation, intermittent fever, and anæmia, are insufficient for the purpose. In order to appreciate this sign, the unaided ear must be applied to some convenient point of the affected side, *e.g.*, over the dull region posteriorly below the angle of the scapula, and the patient directed to whisper some rough word; this will be well conducted to the ear if the fluid be serous, not so if it be purulent. Dr. Bacelli's theory to account for the difference of conduction in the two cases is, that in serous effusions, the fluid, being thin and homogeneous, transmits vibrations with facility, but the more the fluid departs from the homogeneous nature of serum, the thicker it is, and the more clouded by the presence of amorphous protein bodies and formed or corpuscular elements, the less complete the conduction of sound through it. This physical sign is totally different from that of œgophony, which is a compressed lung-sound modified by transmission through a thin layer of fluid.

Unfortunately, although of undoubted usefulness as an additional sign, it cannot be allowed that this sign has the crucial value attributed to it by its distinguished advocate. *Pectoriloquie aphonique* is heard in perfection in some cases of pure sero-fibrinous effusion, but I have also heard it very well marked in some cases of purulent effusion, and perfectly in cases of fœtid sero-purulent effusion. At the International Medical Congress, 1881, I reported ten cases bearing upon this point. "In six of these, in which the fluid was clear, five yielded the sign, the sixth did not. In two cases, also acute, the fluid was purulent; yet in both cases Bacelli's sign was present when one would not have expected it. In two cases, both of old standing, the fluid was serous, containing effete pus elements; in one of these cases the sign was wholly absent, in the other it was present in a modified degree."[1]

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[1] Archivio di Medicina, Roma, 1875. See a critical reference to Dr. Bacelli's paper by Dr. Gueneau de Mussy.—Union Médicale. Jan. 4th, and Feb. 17th, 1876.

There is, indeed, only one criterion in these cases, and that is the exploring puncture made with a grooved needle or fine trochar, and this method may be employed without danger or inconvenience at any period at which absolute certainty of diagnosis becomes of importance.

Pulsation is sometimes communicated to the fluid by the heart's action, and in some cases this pulsation strongly simulates that of aneurysm. Dr. Walshe has referred to this point, and similar cases have been observed by others, in all of which, according to Fraentzel,[2] the fluid has been purulent. In each of the two cases observed by Traube, and also in the one that came under Fraentzel's notice, pericarditis with effusion into the pericardium was present. Traube considers the occurrence of pulsation as due in part to suppurative softening with increased extensibility of the pleura costalis, and in part to the presence of pericardial effusion favoring the transmission of the heart's impulse. Some few years ago Prof. Maclean was kind enough to allow me to examine, at Netley, a soldier who had a very excessive effusion into the left pleura, and who had so marked a pulsation in the left supra-mammary region as to cause some hesitation in performing paracentesis, lest there should be a large aneurysm in addition to the hydro-thorax. A right judgment was, however, without much difficulty arrived at, and a large quantity of *serous* fluid was removed, and with it the doubtful sign of aneurysm disappeared. There was no pericarditis suspected in this case (and the heart was of course carefully auscultated). Another case of effusion, right-sided and of older standing, came under my notice at the Brompton Hospital some months ago, in which a rhythmic impulse conveyed through the fluid suggested aneurysm as a complication. The fluid was, however, removed by several tappings; it was somewhat opaque and at first slightly blood-stained. No aneurysm was present. It is thus clear that neither suppuration in the sac nor pericarditis is necessarily associated with pulsation, and probably nothing more is needed to account for the phenomenon in question than an amount of fluid which shall exercise a certain degree of pressure (neither too much not too little) upon the beating heart. Two or three other instances have come under my observation in which the diagnosis of mere fluid in the pleura has been much shaken by another unusual pressure effect, viz., altered quality of voice and cough. A husky voice, and a laryngeal quality of cough, undistinguishable from that so often heard in cases of mediastinal tumor or aneurysm, have given rise to great doubts as to the diagnosis, yet both phenomena have disappeared after paracentesis.

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[1] "Transactions of the International Medical Congress," 1881, vol. ii., page 146.

[2] Ziemssen's Cyclopædia, vol. iv., p. 638.

Putrid effusions.—Two cases[1] have come under my observation, which lead me to think that sometimes the fluid in sero-fibrinous effusions undergoes spontaneous decomposition, and becomes converted into a fœtid sero-purulent matter, a discolored stinking fluid being removed on paracentesis. On this point a remark of Professor Marshall's is interesting. He observes:—"The sero-fibrinous effusion appears to have a greater tendency to quick decomposition, when air is admitted into the pleural sac, than the sero-purulent or purulent product. Pus is more stable and less inclined to rapid putrefaction than the sero-fibrinous fluid."[2] In the cases referred to, however, there had been no opportunity for contamination from without.

In such cases septic phenomena of the more virulent kind present themselves, of which continued rapidity of pulse, a red tongue, smooth and denuded of epithelium, sometimes aphthous or thinly coated, complete anorexia and vomiting or diarrhœa, are the most characteristic. In the presence of such symptoms we may be sure that an effusion is purulent, and has probably undergone decomposition, and only with its free evacuation and disinfection will they vanish.

In all cases of doubt, then, I would still counsel the timely use of the needle-syringe, by means of which a sample of the fluid can be obtained with little or no pain to the patient, and absolutely without danger of doing mischief.

In suppurative pleurisy the only efficient treatment is by evacuation of the pus, and the progress is, generally speaking, favorable in accordance with the promptness with which the pus is recognized, and its removal effected.

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[1] Viz., Case 7, Trans. International Congress, vol. ii., Medical Section, p. 143, seen with Dr. Stamford of Tunbridge Wells, and a case more recently seen with my colleague, Dr. Finlay, at Middlesex Hospital.

[2] Lancet, vol. i., 1882, p. 300.

CHAPTER VI.

ON PLEURITIC EFFUSIONS CONSIDERED ESPECIALLY WITH REGARD TO OPERATIVE TREATMENT.

PLEURITIC effusions, whatever be their original source or nature, have so much in common with regard to treatment, that they may be conveniently brought together for consideration in this point of view. Much progress has been made in the treatment of these effusions within the last few years, and so much has been written and said upon the subject, that it would take up many chapters, and be quite beyond the practical scope of this work to review the literature in any formal manner. I shall therefore, for the most part, content myself with relating those methods of treatment which have proved most successful within my own observation and experience.

Pleuritic effusions are naturally grouped into:—

(1) Simple inflammatory effusions, in which the fluid is sero-fibrinous and which may be *acute* or *chronic*.

(2) Suppurative effusions, in which the fluid is purulent, and of recent or old date.

(3) Hydro-thorax or dropsy of the pleura.

(4) Hæmo-thorax.

(5) Pneumo-thorax.

1. SIMPLE INFLAMMATORY EFFUSION INTO THE PLEURA.—*Acute effusion.* The general grounds upon which operative interference should be adopted, withheld or delayed, were fully laid down in the preceding chapter. The tendency at the present time is perhaps rather towards too great an anxiety with regard to the presence of fluid effusion, and to a corresponding impatience for its removal.

One might, indeed, feel some misgivings as to whether Nature ought ever to be burdened by having to remove so vast an accumulation, did not experience show that she is fully equal to the task; and when we consider how, throughout the body, effusion and absorption are perpetually going on, constituting an extra-vascular circulation amounting in the aggregate to many pints, we cease to have any real difficulty in understanding this ready removal of fluid collections.

If at the end of a week or ten days from the time when the pleuritic effusion has reached its height, appropriate remedies having been used, there be no signs of any material abatement of the fluid, it will be judicious to draw off a portion of it. Doubtless the employment of purgatives, and diuretics or mercurials would still, in most cases, ultimately effect the withdrawal of the fluid though the natural channels. I have known an acute serous effusion require six months' treatment by tonics and diuretic remedies for its final removal. In another case, after many months of fruitless treatment by ordinary methods, mercury, pushed to salivation, rapidly removed the effusion.[1] But this protracted or severe medicinal treatment is not always successful, and is at least attended with great risk of permanent damage to the lung, and of complication with other diseases. The better course in such cases surely is to adopt the timely removal of a portion at least of the fluid by paracentesis; but the worst course of all is to leave such cases alone or to treat them with indifferent drugs. Dr. Bowditch observes: "I cannot see any valid reason for continuing any active treatment more than one, two or three weeks without puncturing."[2]

In those cases in which there is an indisposition on the part of the vessels and lymphatics to reabsorb the fluid, the effusion is commonly very great, and the intra-thoracic pressure considerable, perhaps equivalent to an inch or an inch and a half of mercury.[3] Now although, during the inflammatory fever, fluid can, even up to this degree of pressure, be effused, owing to the high tension of the general circulation, and the relaxed condition of the vessels locally, yet when the fever has passed, more or less general exhaustion and anæmia prevail, the pressure of the circulation falls, and the effect of the high pressure upon the pleural surface is necessarily to render it especially anæmic, and, by the powerful compression of the lung, to hinder absorption by the lymphatics. The fact, frequently noticed, that after removal of even a small portion of the fluid the rest is rapidly absorbed, almost demonstrates the correctness of this view. Trousseau observes "this greater slowness in the absorption is, perhaps, as much dependent on the pressure exerted by the excessive quantity of fluid upon the serous membrane by which absorption has to be performed, as by the mere greatness of the quantity."[4]

It is probable that absorption of pleuritic effusions takes place largely through the lymphatics, otherwise one would expect to find some albumen in the urine whilst so large a quantity of albuminous fluid was being taken up by the systemic veins. This I have not found, and Dr. Dickinson remarks in his recent work on Albuminuria, "It has been said that the same result (temporary albuminuria) has followed the rapid absorption of serous fluid from the pleura, a sequence, which, to say the least, must be rare."

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[1] Dr. Hope—Medico-Chirurgical Review, 1841—strongly recommended the treatment of pleuritic effusions by mercury.

[2] "Thoracentesis," by Henry J. Bowditch, M.D., 1870, p. 13.

[3] "Observations on Paracentesis," Clin. Soc. Trans., vol. iii., 1870, p. 240, Med. Chir. Trans., vol. lix., p. 187.

[4] Clinical Medicine, New Syd. Soc. Trans. of 3rd edit., 1868, vol. iii., p. 229.

In other cases, on the subsidence of acute symptoms, absorption goes on well enough up to a certain point, beyond which, however, the patient remains at a standstill, his chest being perhaps half full of fluid. In these cases there is usually considerable depression of general health, and iron tonics, good diet, and iodine embrocations or blistering, with change of air, will generally effect complete absorption. If sure that the fluid were not purulent, one would be loth to interfere at this stage.

Chronic pleuritic effusion.—Patients may live for a long time, and with surprisingly little discomfort, with very large effusion into the pleura. Not a few cases are still from time to time met with in which one side of the chest is distended with fluid, the effusion dating from an attack of pleurisy months or even a year or two previously, and which, on removal, proves to be perfectly limpid and serous. The inflammatory attack leading to such an effusion is sometimes only marked by slight symptoms, rapid effusion no doubt relieving the inflammatory condition and certainly removing the pleuritic pains. Trousseau remarks that these patients are frequently quite unconscious of having anything seriously the matter, although the pleura may contain a large quantity of fluid. I have still under my occasional observation a policeman, stalwart-looking enough to frighten thieves, who for four or five years has had his left pleura completely full of slightly turbid serum. After a few partial tappings it became evident that no expansion of the lung could be looked for, and in the absence of any urgent symptoms it has not been thought prudent to submit him to any radical measures of treatment involving long illness and, probably, a permanent thoracic fistula. I think, with Dr. Wilson Fox,[1] that the fear lest the effusion should become purulent from mere lapse of time, urged by Trousseau as a reason for early operation, is not well grounded; there is very little disposition for such a transformation to take place, unless the patient have a fresh inflammatory attack. But perfect recovery in these old-standing cases is well nigh impossible, the lung having become bound down and thickened by long-continued compression, and it is to be hoped that the reproach of their not very infrequent occurrence will soon be removed from us.

I am bound to confess, however, to holding very strongly the belief that there will always be a certain small number of cases in which the lung is from the first coated by an exudative layer of unusual thickness and uniformity, which, as pointed out by Walshe,[2] contracts forcibly and aids the fluid in bringing about a collapse of lung. Such a membranous layer affords a serious impediment to absorption, and is apt to undergo a degree of organization which renders the constriction of the lung a permanent condition. Under these circumstances, and especially in strongly built adult chests (as in the case of the policeman above quoted), a large pleural space remains which it is almost impossible to obliterate, and which must be occupied by some kind of effusion.

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[1] Brit. Med. Jour., Dec., 1877, p. 752.

[2] Diseases of Lungs, 4th edit., p. 252.

Paracentesis thoracis in serous effusions.—Choice of spot for puncture.—Supposing paracentesis to be necessary, the first step to be taken is to select the best position for puncture. The physician is responsible in choosing the site for puncture, and must not share the responsibility with others. In choosing the spot he has to be sure (1) that it is out of reach of diaphragm and heart; (2) that there is no adherent lung there. The *sixth space in the mid-axillary line* is the best point for puncture, and should be selected if, when tested by percussion, palpation, and auscultation, it prove satisfactory. This spot is most convenient because (1) most accessible whilst the patient is reclining in an easy posture; (2) the parietes are here moderately thin, and the intercostal space sufficiently roomy; (3) the mamma in the female is out of the way; (4) we are sufficiently high up to be free from danger of perforating the diaphragm; (5) this point has the advantage over that most commonly chosen, below the angle of the scapula, in there being less probability of the canula becoming blocked by the flocculi, which tend, from the position of the patient, to gravitate towards the back of the chest (most of the dry tappings I have observed have occurred with the posterior puncture); (6) this point has the advantage over one chosen more anteriorly in being more central with regard to the effusion.

Some authors prefer other sites for puncture. The matter is not one of any great importance. Prof. Marshall would choose the anterior weak spot in the chest wall in the fifth space nipple line. Another so-called weak spot, preferred by some operators, is in the line of the angle of the scapula; my point is between them. Of course, in special cases of limited effusion the point for puncture must be selected accordingly, it being remembered, however, that a central rather than the lowest point of the effusion should be chosen for puncture.

Vertical and horizontal marks should be made upon the chest pointing to the precise spot chosen.

Local anæsthesia, puncture.—The patient reclining near the edge of the bed, with the head and shoulders slightly raised and leaning towards the diseased side, a piece of ice with a flat surface, an inch or two square, dipped in salt, should be applied with firm pressure to the spot marked for puncture. In twenty or thirty seconds the spot will be frozen; it should then be rapidly wiped, a small incision made through the skin, and the trochar, previously steeped in carbolic solution (1 in 40), smartly thrust into the pleura. The canula will have been previously connected with three or four feet of tubing filled with water and leading to a vessel containing a certain quantity of water, which must be two or three feet below the level of the patient; or the tubing may be connected with a bottle which can be *gradually* exhausted of air by a suitable syringe. The operation having been completed, the canula must be withdrawn rapidly, the edges of the wound being compressed between the finger and thumb and a fold of lint soaked in collodion applied. A band of strapping three or four inches broad and of sufficient length, should then be firmly applied round the affected side, so as to extend a couple of inches beyond the median line in front and behind.

The following list includes all the apparatus needed for the performance of paracentesis in a case of serous effusion, and suggests some further remarks respecting the performance of the operation.

1. A lump of ice and some salt.
2. A small scalpel or lancet.
3. Carbolic solution (1 in 40).
4. Trochar and canula connected with three feet of tubing; the whole having been previously filled with water.
5. Basin containing one pint of water.
6. Lint. Collodion.
7. Piece of strapping plaster four inches by twenty.

1. The plan named in the text of cutting a plane surface upon a lump of ice, dipping it into some salt, and then applying it firmly to the surface of the skin, is by far the best means of obtaining local anæsthesia for paracentesis thoracis. If no ice be obtainable, the ether spray may be used, or the operation performed without the aid of any anæsthetic.

2. It is always best to make a small incision through the skin (except perhaps when quite capillary instruments are used). The clean-cut wound heals much more readily than the bayonet thrust, and less pain is felt by the patient because the skin is not stretched by the trochar.

3. It would seem needless to add a word of warning on the importance of seeing to the scrupulous cleanness of trochars or aspiration needles used in paracentesis. Cases have, however, come under my own observation, in which carelessness in this respect apparently led to decomposition of the fluid, suppurative pleurisy, and ultimately to the death of the patient.

If an instrument be not fresh from the makers, its purity should be insured by submitting it to the flame of a spirit lamp. If rubbed over first with a little oil, the metal will not be tarnished by the heat.[1]

4. The best instrument for tapping the chest is the simplest one possible which will effect the object required, *viz.*, to remove fluid without admitting air from a cavity with walls more or less elastic, and which is contracting and expanding alternately, the contraction and expansion movements increasing in depth and force as the fluid is withdrawn.

If a simple hydrocele trochar be used, it is inevitable that before any considerable quantity of fluid has been withdrawn, air will be sucked into the chest.

With regard to the question as to the expediency, I should rather say the imperative importance, of avoiding the admission of air during paracentesis in the cases now under consideration, I fail to see the usefulness of any further discussion. True it is that, in some cases, air has been admitted into the pleura without any harmful result, but these cases are exceptional, and against them must be brought other cases far more numerous, in which renewed fever, fresh pleurisy, and the conversion of the serous into purulent effusion has followed.[1] And, apart from these directly evil consequences, let us remember that air is more foreign to the pleural cavity than serum; that whilst air is present it is quite as impossible for the lung to expand, or for the heart to return to its normal position as though fluid were there, nor is the absorption of air by the pleura when altered by recent or old inflammation more easy than that of serous fluid. Why then allow this, at best foreign, and in most cases noxious, element to enter at all? The only plausible answer is, that unless we do so, all the fluid cannot be removed. This is true as a statement, but is no answer, because we do not wish, in any case of simple effusion into the pleura, to withdraw all, or nearly all, the fluid, but only a sufficient quantity to relieve pressure and to encourage the absorption of the remainder.

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[1] For this last practical hint I am indebted to Mr. E. A. Fardon, Resident Medical Officer at Middlesex Hospital.

Hence, then, we must have some addition to the ordinary canula by which, whilst the fluid is allowed to flow from the chest, air is absolutely excluded from entering it.

The simplest contrivance for this purpose is perhaps that ingeniously suggested by M. Reybard, *viz.*, to tie on to the neck of the canula a piece of gold beater's skin rolled into a tube. When wet, this acts as a perfect valve, allowing of the free exit of fluid but collapsing over the orifice of the canula on the slightest aspiration towards the pleura. An ordinary hydrocele trochar can be readily fitted with this form of valve. But a piece of india-rubber tubing attached to the canula, and having its other extremity under water at a lower level than the chest, is the most complete means of excluding air, and we can in this way, moreover, employ whatever degree of syphon power we wish to aid the removal of the fluid.

The trochar which will be found most generally useful for this purpose, is one which I brought before the notice of the Clinical Society in 1870,[2] in the paper already referred to, and I have not since found anything to improve in its construction.

This trochar is, as I was not then aware, essentially the same as that designed by Mr. Charles Thompson, and described in the *Medical Times* in March, 1858. The padding in the cap of the canula through which the stem of the trochar works seems an improvement; it was suggested by Mr. Hawksley, the maker of the instrument.

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[1] The late Dr. Fuller, in an otherwise admirable clinical lecture, was I believe the last clinical teacher who regarded with indifference the admission of air during paracentesis for serous effusions. Brit. Med. Jour., Feb. 3rd, 1872.

[2] *Loc. cit.*

The canula (fig. 11) has a calibre of about $\frac{3}{20}$ of an inch, (4mm.), and is provided with a lateral branch (*a*) furnished with a leather collar on to which the tube piece (*d*) screws. The trochar has only a small and light handle, and works through the cap of the canula; this cap (*c*) is so padded with leather that, when forcibly screwed down, the stem of the trochar is grasped by the padding in an air-tight manner. The tubing attached to *d* should be about three feet long, and should be interrupted, near the canula, by a piece of glass tubing let in so that the nature of the fluid, etc., may be observed. It is a very good plan to fill the tubing, ready attached to the canula, with water before making the puncture. As soon as the instrument has been introduced, and the stilette (*b*) withdrawn, the fluid flows through *a* and no admission of air is possible. The trochar should be dipped in carbolic solution before being used, and the receiving basin lowered so as to allow a syphon column of between two and three feet of tubing.

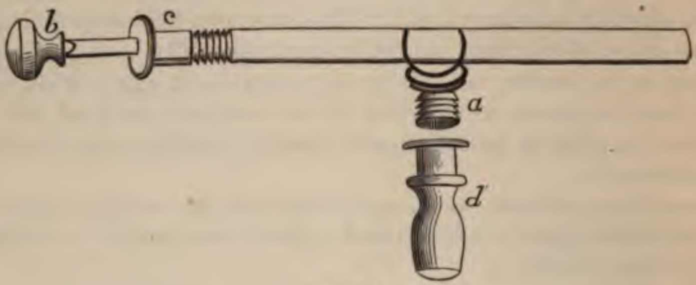

FIG. 11.

It is often convenient, and if properly managed is quite harmless, to withdraw the fluid into a bottle by aspiration. For the purpose now indicated, the fittings of Potain's aspirator are the best. A cork (*c*, fig. 12) made of india-rubber, and of such dimensions as to accurately fit any ordinary wine or seltzer-water bottle, is perforated by two tubes, one of which (*a*) fits on the basin end of the exit tube attached to the canula already described. The second tube *b*, is adapted to an exhausting syringe. Each of these tubes is supplied with a stop-cock, and the tube *d* in connection with the canula should extend somewhat further into the bottle than *b*′.

Now the ordinary "aspiration" method is as follows:—Having fitted the apparatus, to exhaust the air from the bottle, and then having connected the canula-tubing, to open stop-cock *a* when the fluid rapidly fills the bottle. The stop-cock *a* is then again closed, the bottle detached and emptied, and the process repeated. By adopting this method of practice, however, we are employing quite an unknown suction power, which may amount to anything short of 15 lbs. to the square inch, and which is not only quite unnecessary but, if incautiously used, extremely dangerous, and especially so towards the latter part of the operation when there is already negative pressure within the pleura.

A very safe and useful way of using this apparatus is that which I sometimes employ, viz., to open the stop-cock *a*, and just to keep the syringe in sufficient action to permit or encourage the flow of fluid through the exit tube *d* into the bottle. When the bottle is filled, *the stop-cock, a, must be closed* before the cork is withdrawn, otherwise there will be a rush of air into the chest. I cannot say that this plan has any advantage over the syphon, both methods are very convenient and safe for removing fluid from the chest, the syphon excelling the other in the uniformity and measurability of the power employed, and also in simplicity and freedom from extra fittings. If a soft plug of fibrine block the canula it may be impelled through the tube by a momentarily increased aspiration power, or it may be dislodged by reversing the syphon or cautiously thrusting back the trochar.

In cases in which fine capillary trochars are used aspiration is necessary, and the usual method of employing this agency does not remove the fluid with the same dangerous rapidity as through larger instruments, but there is the same uncertainty as to the amount of negative pressure induced within the thorax. In some cases, however, in which there is doubt respecting the diagnosis, a fine exploratory trochar, previously passed through the flame of a spirit-lamp, should be first employed.[1]

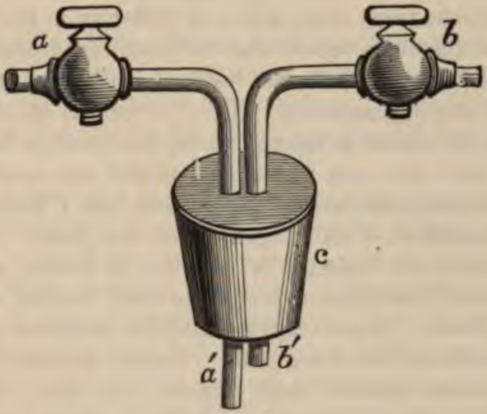

FIG. 12.

The usual plan of applying a bandage round the chest after paracentesis is not a good one; it hampers the movements of the healthy side and distresses the patient. It may be a question whether any application of the kind is necessary, but I have always preferred to keep the affected side at rest by the firm application of a band of strapping round the lower ribs to beyond the median line in front and behind, thus leaving the healthy side free.

In acute pleuritic effusions the operation of paracentesis has rarely to be repeated, the remaining fluid being absorbed without much difficulty.

In cases, however, of what may be called *chronic hydro-thorax* (cases which will become more rare as acute pleuritic effusion is more efficiently treated), in which the lung is bound down by adhesions more or less strong, repeated tappings are necessary, and they should be performed in the way above described and with the same scrupulous care to avoid admitting air. The syphon power employed may sometimes, in these cases, be increased with the view of aiding lung expansion. In most cases the lung partially expands, and is partly met by some flattening of the chest wall, attraction of the heart, and enlargement of the opposite lung. Meanwhile no symptoms arise from the operation, and after a rest of a day or two it is better to allow the patient to be about and to enjoy the fresh air. Tonics and iodine frictions may be employed in the intervals, and a pad of spongio-piline may be inserted underneath the strapping in the infra-mammary region, so as to keep up some pressure at this portion of the chest where flattening first commences.

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[1] It would be very easy of course by a pressure guage fitted into the bottle to know at any moment what degree of aspiration is being used, but this arrangement would require a special bottle, whereas a great advantage of Potain's cork adjustment is that it renders any ordinary bottle available.

2. EMPYEMA.—Cases of suppurative pleurisy resulting in pyo-thorax are necessarily of far graver character than those we have just been considering, and present many more points of difficulty in their treatment.

There is good reason to believe that in some rare instances, especially in children, the purulent fluid has been absorbed, the pus cells having first undergone fatty degeneration.[1] Dr. Fox regards M. Moutard-Martin (*De la Pleurésie purulente*) as having placed this question beyond dispute. I have myself seen one case which has satisfied my mind as to the possibility of a local empyema becoming absorbed, but I know not the ultimate fate of the patient, a child aged about five years. Dr. Edebohls[2] has recently related an instance in a child, in which, after proof by needle puncture of the effusion being purulent further operative treatment was not allowed. Some months later, the child was again examined and its chest was found free from fluid. Twelve months after, however, the signs of decided phthisis were present. In some other cases the fluid portion of the effusion is absorbed, and the remainder thus inspissated is deposited in a thick layer on the pleura. The spontaneous disappearance of such effusions is, however, too uncommon to be expected; and the process of reabsorption is one too full of peril to be anticipated with anything but dread. It is indeed an attempt at such absorption that occasions the most characteristic hectic symptoms in the second stage of suppurative empyema. In the event of the more liquid portions becoming absorbed and inspissated pus left behind, although for a time the patient may do well, yet at any future period pleuritic abscess or secondary tuberculosis may ensue upon the softening of the caseous deposit.

If left to run its natural course, a case of purulent effusion will in the infinite majority of instances either (1) prove fatal from exhaustion or syncope; or (2) after a few weeks of hectic, the empyema may suddenly burst through the lung with perhaps immediately disastrous results. More commonly, however, this sudden outburst is followed by long-continued profuse expectoration of decomposing purulent matter with symptoms of chronic septic poisoning, and gradual exhaustion with secondary disease of the lung. Cases have occurred in which an empyema has burst into the peritoneal cavity, or the sheath of the psoas muscle, in other cases it may simulate peri-nephritic abscess; or (3) the pus may point and discharge by a fistulous opening through an intercostal space. It is contrary to all the principles of enlightened treatment passively to allow either of these phenomena to occur, and we can never do so without taking great blame to ourselves when it is too late. In no other situation in the body would a large collection of pus be allowed to remain after it had been fairly recognized, and we should not depart from one of the best-established of all rules in medicine because statistics bring out the fact that the results of paracentesis in empyema are still far from satisfactory.[1]

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[1] Dr. Wilson Fox, Brit. Med. Jour., Vol. 2, 1877, p. 752; Dr. Clifford Allbutt, idem p. 726, and Dr. Goodhart, p. 795.

[2] New York Medical Record, Jan. 26th, 1884.

It is perfectly clear, then, that we must adopt some surgical measures for the treatment of all cases of empyema, or take upon ourselves the responsibility for a large mortality; fortunately our hands have been greatly strengthened in this matter by the advance in surgery within the last few years.

It is still more true with suppurative than with serous effusions, that the greater the duration of the effusion, the more difficult is its ultimate removal, *because in the former cases we must evacuate all the pus from the pleura*, and the more complete the power retained by the surrounding walls of closing in and obliterating the cavity left behind, the better. Hence, in active cases, as soon as it is ascertained that there is pus in the pleural cavity, the question of its removal should be at once considered.

I have already referred to the signs upon which the diagnosis of suppurative as distinguished from serous pleurisy may be made, (see page 65). The association of the attack with pyæmia, scarlatina, the puerperal state, typhoid fever, the scrofulous diathesis, or already existing pulmonary disease, renders it *much more probable* that the fluid effused will be purulent. A typhoid character of symptoms, with great rapidity of pulse; daily elevations of temperature, continued beyond the period at which in simple pleurisy it should subside; continued rapidity of pulse; the occurrence of hectic sweats and of *secondary chills;* marked emaciation; anorexia and furred tongue are all signs which strongly point to pus in the pleura. Œdema of the side sometimes appears as a later sign, and when it occurs is of great importance.

It is impossible to lay down any hard and fast rule as to the exact time at which all such cases should be interfered with. No two cases are precisely alike, and each must therefore be dealt with on its own merits and in the discretion of the observer. But the general rule laid down by Dr. Hamilton Roe, who (with Hughes and Cock of Guy's Hospital and Trousseau in Paris) practically revived the operation of paracentesis thoracis, should be acted upon, viz., "that as soon as it is clear that pleurisy is subdued, and that a large quantity of fluid remains in the chest, we should proceed at once to ascertain its quality, by introducing the exploring needle[1] (invented by Sir B. Brodie), and if it is found to be purulent, the operation should forthwith be performed."[2] The period for exploration here referred to by Dr. Roe would be from about the second to the third week of the disease.

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[1] See cases related by Dr. Goodhart in Guy's Hospital Reports, 1877, p. 183.

The next question is as to the best method of removing the fluid. It may be convenient, and particularly so in cases in which symptoms of asphyxia or syncope are threatening, to remove in the first instance a portion of the pus by means of the syphon or aspirator; and some authors prefer, in children at all events, to try one or two aspirations before having resort to the more radical operation. Although by this means temporary relief is obtained from any pressure-symptoms that may be urgent, the effusion will certainly re-accumulate, and we cannot be said to have yet dealt with the case until the pus has been thoroughly evacuated. Dr. Clifford Allbutt's objections to incomplete tappings or partial aspirations in purulent effusions, seem to me to be very valid ones. He remarks,[3] "My two objections, and these complete ones, to repeated aspirations are: —1. Aspiration does not prevent the formation of a pulmonary fistula. 2. It does not prevent absorption, but rather favors it. By the pressure of a full cavity, absorption is often prevented and fever absent; draw off some of the pus, you relieve pressure, and absorption begins." These objections hold good in acute purulent effusions. In chronic empyema of old standing the conditions present are different.

There is this very important difference then, with regard to treatment, between serous and purulent effusions, that a serous effusion, not being in itself hurtful, need only be partially evacuated when circumstances require it; a purulent effusion on the other hand is of itself highly poisonous, and, although it may be convenient for the moment to remove a portion only, yet no real progress will have been made in the treatment of the case until the whole of the pus has been evacuated, and free exit provided to prevent any further accumulation. The objects in view in paracentesis for serous and for purulent fluids are very different. In empyema we desire completely to evacuate the pus, to allow as little subsequent suppuration as possible, and to provide free drainage for such pus as may form.

It is clear that we cannot empty the pleura, even in recent cases, without admitting air in place of the fluid. Hence we must adopt one of three measures. 1. Either to disinfect the air admitted into the pleura, and having inserted a drainage tube, to close the wound with antiseptic dressings, to be renewed with the same precautions every three or four days.

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[1] The ordinary subcutaneous injection syringe (I think first recommended for this purpose by Dr. Ringer) or the needle of an aspirator would now be substituted for the old-fashioned grooved needle.

[2] Medico-Chirurgical Transactions, vol. xxvii., p. 198.

[3] British Medical Journal, Nov. 24, 1877, p. 727.

2. To make a single free opening and insert a tube through which the pleura can be daily washed out with some disinfecting fluid.

3. To make a double opening and introduce a drainage tube, so as to permit of the escape of pus as rapidly as it is formed.

The point to be chosen for incision in cases of purulent effusion is a different one from that to be preferred in serous effusion. A moderately low opening is desirable, in the seventh or eighth intercostal space, and *in the posterior axillary line*, due precautions being taken with regard to position of heart, lung and diaphragm. My reason for recommending a lower and more posterior point for puncture in these cases is, that in acute empyema we wish to empty the pleural cavity of pus, and we look for obliteration of the abscess-sac by the descent of the lung as it re-expands, and by the return of the heart to its normal position: these processes converge towards the lower and postero-lateral position of the chest.

Professor Marshall[1] would lay down the rule never to perform open paracentesis lower than the sixth or seventh interspace at the sides of the chest, and in the adult, he recommends in preference the fifth space in the nipple line over the "weak spot" of the chest wall. Further observations are needed to determine the relative advantages of different positions of opening in cases of acute empyema, and probably the results will show that there is room for differences of opinion. If there be any doubt at the time of operating as to the eligibility of the spot selected for puncture, an exploratory trochar should be first introduced.

Antiseptic operation.—Of the three above-named methods, the first is to be preferred, and it is on the whole the least troublesome, since, although there are several points of detail needing attention during the operation and at the subsequent dressings, these dressings require to be removed at less frequent intervals, and all injections of the pleura are dispensed with.

I will briefly give the headings of this method, which the practitioner will have no difficulty in supplementing as soon as the principal objects in view are clearly discerned.

(*a*) The apparatus required, consists of—1. A Lister's hand-ball spray producer or small steam-spray apparatus charged with carbolic acid solution (1-40), and having pieces of linen wrapped round the submerged ends of the tubes so as to prevent them from becoming blocked by any foreign particles.

2. A tray containing a solution of carbolic acid (1-20) in which to

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[1] Lecture on Diseases of the Chest Cavity, delivered at the Brompton Hospital. Lancet, March 4th, 1882.

place all instruments to be used, which instruments must of course be thoroughly clean.

3. The instruments that should be at hand consist of a bistoury, a full-sized trochar and a fine exploring trochar, drainage tubing (well soaked in antiseptic), scissors, dressing and torsion forceps, and in case of removal of rib, raspatory, bone-clippers, etc.

4. An anæsthetic must be administered or local anæsthesia employed. It is most convenient to give chloroform and ether, and these anæsthetics are as a rule remarkably well borne by empyema patients.

5. The hands of the operator and assistants must be carefully washed and rinsed in carbolic acid solution, and the nails cleansed; the same precautions being observed each time the case is subsequently dressed. The surface of the patient's chest must be well sponged over with carbolic lotion 1 in 40.

6. Boracic or carbolized charpie. Antiseptic gauze, not less than eight layers thick, and about 12 by 16 inches area. Under the outermost layer of the gauze, a piece of thin prepared mackintosh must be inserted, care being taken that it does not reach beyond the edges of the gauze in any direction. Some rollers for bandaging made of the same antiseptic gauze material must be ready to keep the application in place. And a turn or two of elastic bandage for the purpose of finally maintaining in close apposition the free margins of the dressings.

After a preliminary incision, which should be sufficiently free (not a mere nick, as for the trochar in serous effusion), down to the intercostal membrane, either a large trochar should be inserted or the incision continued with the knife.

An incision is apt to get occluded by false membrane, and in this respect the trochar is preferablè; but circumstances at the time, such as the amount of intercostal space, will determine the employment of one or other instrument. Some surgeons prefer, even in acute cases, to resect an inch or so of a rib, with the view of getting more room. I would not recommend this procedure, at least in the first instance. In a case in which difficulties have arisen at the first tapping, and in which there is reason to suspect loculation of the empyema, it is a great advantage to resect a rib, and thus get room for the free introduction of the finger, to break down adhesion cysts. The escape of fluid through an incision may be facilitated by introducing through the incision the blades of a Trousseau's dilator, or of clean ordinary dressing forceps, and opening them out so as to stretch the wound. As much of the fluid having escaped as will do so, a piece of drainage tube three to five inches long, is introduced, through which probably more fluid will at once escape. The side having been washed, a piece of protective is placed round the tube, which, unless provided with a broad rim, must be retained by being transfixed by a silk suture secured round strips of plaster to the side of the chest. A broad rim to the tube is safer however. A soft thick pad of charpie is then to be placed over the orifice of the tube, and the broad eight-layer thickness of antiseptic gauze superposed and kept firmly in place by many turns of carbolized bandage extending above and below, so as to envelope the whole chest. Some arrangement of shoulder straps will prevent the bandages from slipping down, and a few turns of elastic bandage are very useful to preserve the free edges of the dressing in close contact with the chest. The air, except that first introduced under the spray, and what may filter through the thickness of the dressings is thus excluded.

It is often necessary to dress the wound again, with precisely the same precautions, a few hours later, the dressings having become soaked with discharges; one full day may then elapse, and subsequently perhaps two or three days. The tube should be removed and washed on the third, and subsequent, dressings of the wound.

The patient must be kept in bed, and the strength well supported by nutritious and digestible food. The temperature must be carefully watched, and will be found, if all has gone well, to fall to the normal.

It is very important before commencing each dressing to see that the spray producer is properly charged, and in good working order; any carelessness in detail will neutralize the results of all former trouble. The discharges upon the removed dressings should be perfectly free from odor, and after the first two or three changes, become very small in amount. Thus, the pleural sac is kept practically empty, and the entry of air being more ready into the lung, than through the dressings, the gradual expansion of the lung is encouraged.

The drainage tube will require shortening as the cavity contracts, and the lung expands, and will by degrees be removed altogether.

The above is undoubtedly the most successful method of treating cases of acute and subacute empyema, and although it seems more troublesome than less thorough methods, yet it takes really very little more time, and the occasions for changing the dressings are less frequent.[1] The fact that under this system, the dressings after the first few days may be left undisturbed for two or three days together, is of obvious advantage to the patient by keeping the cavity for these intervals practically closed, and thus affording the lung the very best opportunity of re-expanding. I am far, however, from contending that the antiseptic method is the only one that can with safety be adopted. I say that it is the best and most favorable to the rapid recovery of the patient. .

Single opening and injection of pleura.—Supposing the antiseptic treatment not to be strictly followed, all instruments and tubes used should still be steeped in carbolic lotion. The lighter and simpler the dressings used, the better. A large piece of absorbent cotton-wool should be first laid over the tube for the discharge to soak into, and a pad of oakum applied over that, the whole being kept in place by a few turns of elastic bandage round the chest. For the first two or three days the dressings will have to be changed twice daily; then, if the drainage be perfectly free, the discharge will readily diminish and remain sweet, and one dressing daily will be sufficient. The drainage-tube must be removed at least once every two or three days, and thoroughly cleansed or renewed. The temperature of the patient must be carefully observed; it is an important test of the efficiency of the treatment. If there be any decided rise of temperature we may fear that some accumulation of pus is present. Unless the contents of the pleura be fœtid, antiseptic injections are best avoided, since they tend to retard the expansion of the lung. In those cases in which they are necessary they should be used with care. The pleura should never be *syringed out*, but an irrigator should in all cases be used. A tin pot or glass vessel, provided with a hole near the bottom, to which tubing is fitted, the tubing being supplied with a nozzle or pipe for insertion into the wound is the simplest and best irrigator, or a syphon arrangement may be extemporized. The vessel, having been filled with the lotion required, is simply raised a foot or two above the patient, when the fluid flows into the chest equably, and with a uniform and measurable pressure. As it escapes from the chest the fluid is caught by a tray or other suitable receiver. The irrigator and syphon have the advantage over the syringe, inasmuch as with them we know exactly how much force we are using, and this force is used equably and slowly. Amongst the sudden deaths that have occurred after paracentesis, not a few have happened whilst the pleura was being washed out. Syncope and cerebral embolism are the most common causes of such deaths; the one likely to be induced by any sudden increase or removal of intra-thoracic pressure, the other by such similar disturbance of pressure as may loosen any clots which have formed in the veins of the compressed lung. Amongst the best disinfecting lotions may be mentioned—iodine, one drachm of the tincture to the pint of warm water; carbolic acid, two per cent.; Condy and water; quinine solution, two or three grains to the ounce; boroglyceride has recently been used in watery solution, one to forty. In the case of very young children, who are terrified at injections, immersion in a warm bath colored with Condy's fluid is an ingenious plan suggested by Dr. Barlow and Mr. Parker.

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[1] The materials are sold by Gardiner of Edinburgh, Mayer and Meltzer of Great Portland Street, and Krohne and Sesemann of Duke Street, London.

It is a question likely to be more fully debated, whether, in acute cases of empyema, resection of a portion of a rib be desirable. The operation is a very simple one, and has been advocated at this stage of the disease by Mr. Godlee.[1] Should the tube become nipped this operation may at any time be performed, but in the first instance, *in acute cases*, it is rarely necessary or advantageous.

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[1] Proceedings of the Roy. Med. and Chir. Society, N.S., vol. i., p. 255.

Drainage by double opening.—This method of treatment, which consists in making two openings, and passing a drainage tube through the chest cavity from one to the other, is but very rarely practised now for cases of acute, or indeed in chronic, empyema. It would be quite unjustifiable to adopt the method as a primary measure. For cases treated antiseptically one opening is quite sufficient as a rule.

This plan of drainage may, however, be resorted to on the failure of one of the other methods. A long probe, or curved steel sound provided with a bulb at one end, is inserted either into the opening already present, or through a fresh incision made at one of the anterior interspaces, and passed directly backwards and downwards so as to impinge posteriorly opposite an interspace. The surgeon then cuts down upon the bulbous end of the sound, and having secured to it an india-rubber drainage tube, withdraws the sound, carrying the tubing with it through the chest. The two ends of the tube are then secured outside the chest in the usual way. By this means, as fast as fluid forms in the pleural cavity it is drained off.

In some cases in which we cannot hope for, or do not desire, the lung to expand, this is one of the best methods of treatment; but in all tolerably recent cases the expansion of the lung *is* one of the main objects of our solicitude, and other methods are therefore preferable. In an empyema which proves to be loculated, this plan of treatment is especially suggested, but probably the more modern method of resecting portions of one or two ribs, and with the finger or a sound freely breaking down the adhesions, is far more efficient.

The treatment of *chronic empyema* of old standing is a question of great difficulty, the cases which occur being so various in their nature and origin, and also in their duration and in the degree of expansibility of the lung. They may be divided into—

1. Cases of simple chronic empyema.
2. Cases in which the empyema is secondary to some lung disease, most commonly phthisis.
3. Cases in which the empyema has supervened upon pneumo-thorax.
4. Cases in which the empyema is associated with bronchial fistula or external sinus.

It is to be hoped that, as time goes on, and effusions into the pleura are more promptly treated in the acute stages, cases of simple chronic empyema will be less frequently met with.

The degree in which the fluid is purulent varies very much. In some cases it is greenish and opalescent, in others opaque and creamy looking. In very few cases of old standing can the pus be regarded as active, the corpuscles being as a rule dead and more or less fattily changed. It is, however, much more difficult to deal with this purulent fluid than with serum, and active suppuration is also more readily set up in a pleura which has already yielded pus.

In these old-standing cases, we can no longer hope to gain more than a very partial re-expansion of the compressed lung. Our treatment is directed to secure the removal of the fluid, and obliteration of the pleural cavity, the imperfect expansion of the lung being met by flattening of the chest-wall and enlargement of the opposite lung. Enlargement and increased function of the healthy lung is indeed the final aim of our treatment.

These results can only be obtained by prolonged and steady treatment. Two or three successive tappings with the trochar and syphon may be employed, with the object of withdrawing sufficient fluid to induce a negative pressure within the thorax, and thus to encourage expansion of lung, flattening of chest-wall, and encroachment of neighboring organs. In the intervals of the tappings, the chest should be strapped round its lower part, and a compress applied to the infra-mammary region, the part where flattening first commences. We may, by this means, cause the lung at its upper part to contract adhesions to the chest wall, the gradual growth downward of which obliterates inch by inch the pleural cavity. The resonance of the opposite lung meanwhile gradually extends across the sternum, and the heart is drawn over with it.

Our degree of success in these cases depends mainly upon their duration. If, after a fair trial, we do not seem to be making further progress, a double opening and the insertion of a drainage tube is one plan left to us. Another is a free incision and complete evacuation of the purulent fluid under the carbolized spray, and the subsequent antiseptic treatment of the wound. A third is the resection of three or four ribs, in conjunction with a free opening, by which means the contraction of the chest wall is facilitated. Professor Marshall has suggested the division subcutaneously of the cartilages of some of the ribs with the same view.[1]

The spot to be chosen for incision in cases of chronic empyema in which the lung is presumably firmly bound down, should be at a higher point than in recent cases, since the cavity will be closed, less perhaps by the descent of the lung than by the sinking in of the chest wall, the coming over of the heart and the raising of the diaphragm. Prof. Marshall well points out that if a low interspace be chosen the tube will soon be deflected upwards, and interfere with the apposition of the diaphragm and thoracic wall. Should this inconvenience arise at any period of the treatment it will be best to make a second opening higher and allow the lower one to close.

I have just sufficiently alluded to the methods of dealing with *simple*

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[1] Lancet, March 11th, 1882, p. 381.

chronic empyema, and looking to the great difficulties that are in the way of effecting a cure in these cases, the long illness their treatment involves, and their frequent termination in chronic thoracic fistula, and finally lardaceous degeneration of organs, we may well pause in cases which present no active symptoms, and consider whether they are not best left alone. Each case must be judged of on its own merits, the presence of hectic or of undue pressure upon the heart or the opposite lung being symptoms especially calling for interference.

In cases of empyema secondary to lung disease, and especially in those that have resulted from pneumo-thorax, and in which the air has been gradually replaced by fluid, the rules of treatment would be somewhat different from those guiding us in simple chronic empyema. In the first place we should be in no hurry to interfere. One may not infrequently note an amelioration of symptoms, sometimes even a cessation of cough and expectoration in cases of phthisis, in which pneumo-thorax has occurred on the side of advanced disease; and in such cases it is well to leave the lung compressed for a while by fluid, to give time for the activity of disease to subside, and for the perforation of the lung to close, which does happen in a fair number of cases. The time probably comes sooner or later when interference is necessary, on account of the supervention either of important pressure signs or of septic phenomena.

The point now is to evacuate the pleura without bringing to bear any negative pressure upon the lung, which we desire still to keep collapsed and at rest. A free opening, with irrigation of the pleura, is therefore indicated.

In a third class of cases in which we have to deal with an empyema that has burst through the lung, and established a bronchial fistula, free external incision is also to be advocated, but even although the expectoration be fœtid it is well to attempt antiseptic treatment; for the lung may not be materially diseased, and provided the pleura be kept drained, there is good reason to hope that the perforation may granulate and become closed, and the lung again expand. Hence it is better, if possible, to get the discharges sweet by efficient drainage and antiseptic measures, rather than by injections; this can in some cases be effected.

Cases of local empyema require incision, and, as for more general collections, the seat of incision should be as nearly as possible opposite the centre of the empyema.

CHAPTER VI.

PNEUMO-THORAX—HYDRO- AND PYO-PNEUMO-THORAX.

PNEUMO-THORAX has since the time of Laennec,[1] who first rendered it clinically recognizable, attracted considerable attention, especially amongst French and English writers. Laennec described pneumo-thorax as of three kinds, viz., (1) simple, (2) associated with fluid effusion into the pleura, and (3) with fluid effusion and a fistulous opening communicating with a bronchus. In the first two non-perforative varieties of pneumo-thorax, the gas found in the pleura was, Laennec affirmed, effused there by simple exhalation from the pleura (or the fluid it contained), or it was the product of decomposition. In the third or perforative variety the air gained access to the pleura through the fistulous opening in the lung. In an able article in the *Gazette Hebdomadaire* for 1864, M. Jaccoud maintains that there is no sufficient proof of the occurrence of simple pneumo-thorax, and expresses serious doubts as to the occurrence of air in the pleura at all, save occasionally perhaps from the decomposition of liquid effusion. M. Jaccoud contends that the cases of so-called simple or essential pneumo-thorax, have probably been cases in which minute perforation of the pleura has occurred, the aperture having soon closed, and, in some instances, the air subsequently undergone absorption. There can be little doubt that M. Jaccoud's criticism is just, and that although in gangrenous and some allied conditions of the lungs or pleuræ, some gaseous products of decomposition may collect in the pleural cavity, or the air may, as suggested by Dr. Gairdner, transude through the more or less devitalized membrane, yet such cases are uncommon, and are moreover rendered unworthy of notice beside the gravity of the lesions which have given rise to them.

The causes of pneumo-thorax may, therefore, be thus enumerated. (1) Perforation of the pleura by the breaking down of sub-pleural nodules in the course of phthisis. (2) The rupture of air vesicles in emphysema or during great expiratory efforts; the separation of sloughs in gangrene, or the breaking through the pleura of an hydatid cyst or a pulmonary hemorrhage. (3) The discharge of an empyema through the lung or the thoracic wall. (4) Gunshot and other wounds of the thorax. Excluding gunshot wounds, the discharge of empyemata and gangrene of the lung, it is infinitely rare for pneumo-thorax to occur from any other cause than the breaking down of sub-pleural consolidations in phthisis.

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[1] Traité de l'Auscultation, t. ii., p. 240 et seq.

About five per cent. of cases of phthisis die with pneumo-thorax.[1] The constant breaking down of tubercular and pneumonic infiltration in the course of this disease tends, by undermining the pleura and interrupting its vascular supply at certain points, to cause it to soften and to give way during some trifling increase of air pressure from cough or effort. The perforation may occur at any period of phthisis: even at the very commencement of the disease a small sub-pleural tubercle may soften and break through the pleura, but such an occurrence is much more common in the more advanced stages.

In the majority of cases of phthisis, as already pointed out, pleurisy of a dry and adhesive kind accompanies and keeps pace with the progressing lung consolidation. But it may be observed *post mortem* in some cases which are marked by acuteness of process, and by the pneumonic character of the consolidations, that no adhesion between the pleural surfaces has taken place; each pleural surface, the visceral pleura more especially, being covered by a thin, finely granular, semi-transparent layer of lymph, which may readily be scraped off with the knife, leaving the shining, almost healthy-looking pleura denuded. At certain points of the surface thus affected will be seen opaque yellow spots, some close under the pleura, others more dimly seen through it, varying in size from that of a pin's head to a split bean, on making a vertical section through any of which, we divide a more or less softened caseous nodule, having its centre perhaps already excavated. It is at some one or more of these points that the pleura most commonly gives way.

Pneumo-thorax is thus most likely to happen in the more acute pneumonic forms of phthisis. It is least apt to occur in the more chronic fibroid forms of the disease. In these latter varieties of phthisis, however, the complication is sometimes met with. In some papers on this subject contributed to the *Medical Times and Gazette*, for January and February, 1869, I pointed out that sinuses, in no respect differing from those one sees extending from chronic abscesses, or carious bone, may sometimes be found extending from old cavities towards the surface. Such sinuses may penetrate both layers of the pleura and open into the subcutaneous cellular tissue of the thoracic wall; or if, as is rarely the case, the pleura be not firmly adherent, a communication with the pleural cavity is established. In one remarkable case described in the paper referred to, a sinus had opened from an old cavity through the posterior mediastinum into the opposite pleural cavity. When a communication is thus effected between a more or less deep-seated cavity and the pleural surface, the perforation is so oblique or sinuous as to be always practically valvular.

Pneumo-thorax occurs somewhat more frequently on the left than on the right side, although the experience of different authors varies on this point.[1]

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[1] A contribution to the pathology of pneumo-thorax, by Samuel West, M.D., Lancet, vol. i., 1884, p. 791.

The gas effused into the cavity of the pleura approximates in composition to that of expired air. Dr. Davy[2] found 100 parts to consist of carbonic acid 8 parts, and of nitrogen 92 parts. In five examinations of the gas from a case of pyo-pneumo-thorax at different periods, he found it to vary in composition, the highest proportion of carbonic acid being 16, oxygen 1.5, and nitrogen 82.5, the lowest, carbonic acid 6.0, oxygen 5.5, nitrogen 88.5. Dr. Duncan[3] estimated the fœtid gas from a case of pneumo-thorax secondary to empyema to contain 26 parts of sulphuretted hydrogen and carbonic acid, and 74 nitrogen.

Whether the communication with the pleura be patent or valvular is a matter of some importance as regards both symptoms and treatment. (*a*) If the opening be direct and patent, there can be no positive air-pressure within the pleura since no air can be pent up there. By means of a trochar fitted by tubing to a water-pressure guage, I have ascertained *post mortem* the degree of intra-pleural pressure present in 16 cases of pneumo-thorax. In 4 out of these cases the pressure was *nil.* (*b*) If the opening be oblique or valvular, although during inspiration air may enter the pleura, yet the moment the air is compressed in expiration the sides of the oblique opening close, or the tongue of the valve is depressed and the air cannot escape at all, or only with more or less difficulty according to the perfectness of the valve. In 12 cases out of the 16 above mentioned, there was more or less intra-pleural pressure present varying in degree from 1¾ inch to 7 inches of water.[4]

The *first* effect of perforation of the pleura is effusion of air, and, perhaps, the escape of some purulent fluid from the ruptured lung into the pleural cavity; the *second* effect is more or less intense pleuritis; and *thirdly,* if the patient survive, we usually get more or less effusion, generally purulent in character.

The principal *symptoms* of pneumo-thorax are—sudden acute pain in the side, followed, or rather attended, by great dyspnœa and shock. The pulse becomes frequent, feeble, small, the respirations relatively more frequent than the pulse, and the voice feeble or suppressed. There is occasionally great hyper-æsthesia of the affected side. The position of the patient is frequently changed, he may sit up or recline, with the head raised and an inclination to the sound side, (after fluid effusion to the affected side); the sitting posture with slight inclination forwards, and with the elbows resting upon the knees, is the one most commonly chosen. There is nothing absolutely characteristic about these symptoms, except, perhaps, the suddenness with which they may supervene. The patient is sometimes conscious of "something having given away," and feels a peculiar trickling, cold sensation, associated with the pain in the affected side. All the symptoms of pneumo-thorax may be most closely simulated in an attack of acute pulmonary congestion supervening upon already advanced disease. On the other hand, there may be an almost entire absence of any special symptoms to mark the onset of the attack. In cases in which the pneumo-thorax supervenes in an advanced stage of the disease, "palpitation" may be the chief complaint of the patient.

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[1] Louis, eight cases, seven left-sided; Walshe (collected) eighty-five cases, fifty-five left-sided; the author, thirty-nine cases, twenty-three left-sided (Diseases of Lungs, &c., 1878, pp. 125 and 142); Samuel West, eighty-three cases, forty-two left-sided. Wintrich, Regnaud, Lebert and Weil observe left-sided cases to be twice as frequent as right. Laennec regards the right side as most frequently affected (quoted by Samuel West, Lancet, vol. i., 1884, p. 791). The point is of little practical importance.

[2] Philosophical Trans., 1823-24.

[3] Edin. Med. and Surg. Journ., vol. xxviii., p. 827.

[4] Medico-Chir. Trans., vol. lix., 1876, p. 179.

The *physical signs*, however, of pneumo-thorax are very positive, and can rarely be mistaken for those of any other disease. The alarmed, anxious and distressed countenance, the evident urgency of the dyspnœa usually amounting to orthopnœa, and the small, whispering voice are in themselves, in marked cases, strikingly characteristic. I will enumerate the chief physcial signs, and will dwell only upon those which are most essentially important.—The affected side is enlarged, the shoulder raised, the intercostal spaces effaced, and little or no movement is perceptible with respiration, whilst the opposite side labors with the rapid breathing, its soft parts receding with each inspiration. The heart is displaced towards the sound side, and the abdominal organs are depressed. The percussion note is greatly hyper-resonant or truly tympanitic, where it had perhaps before been dull. Respiratory murmur is either absent, or very feeble; and at one or two points more or less distant amphoric breathing may be heard with metallic whisper and echo on coughing. Pectoriloquy is scarcely ever present. Metallic tinkling may be heard, especially after cough. Vocal fremitus is either absent or much diminished. On applying the stethoscope over one point of the area affected, whilst at another point percussion is made, using coins or other hard substances for the purpose, a peculiar ringing sound is heard (*bruit d' airain*) very characteristic of pneumo-thorax.

Later, when effusion of fluid has occurred, there is dullness at the lower, and hyper-resonance at the upper, part of the affected side, the distribution of dullness and resonance shifting with the altered position of the patient. On placing the ear down upon the chest, and giving the patient a somewhat abrupt shake, a splashing sound may be distinctly heard, perfectly characteristic of hydro-pneumo-thorax. This "succussion" sign may be observed in cases in which we can find no other evidence of the presence of fluid in the pleura. The explanation of this is simple enough; the moment pneumo-thorax occurs, the diaphragm on the affected side becomes flaccid and more or less concave, drawn downwards by the weight of the abdominal organs, and in its concavity a certain quantity of fluid may collect without yielding percussion dullness. In cases in which there is a considerable quantity of fluid present, intercostal fluctuation may be made out; or, on sharply percussing immediately below the line of contact of resonance and dullness, a thrill may sometimes be detected by the finger, significant of fluid vibrations.

Hyper-resonance, absent, feeble, or *amphoric* respiration sounds, and *displacement of heart* are, however, the signs of pneumo-thorax of central importance about which the other signs of less value may be grouped. These three signs alone are sufficient to render the diagnosis certain, and their presence can be ascertained by a physical examination which will not add to the distress of the patient.

The degree and extent of the *hyper-resonance* depend upon the quantity and tension of the air that has escaped into the pleural cavity. In some cases adhesions are so strong and extensive as to limit the pneumo-thorax to a small portion only, usually the base, of the pleural cavity. In such cases the symptoms are rarely urgent, and their cause may escape notice. But as a rule the whole lung is collapsed save, perhaps, at the summit, where there are frequently some old adhesions. The hyper-resonance has a drum-like quality which is usually characteristic, but in cases in which the tension of the air is very great, the vibrations of the chest-wall are less free and the tympanitic note somewhat deadened. The boundaries of hyper-resonance include the sternum, and may extend beyond it towards the healthy side, and if the left side be affected the normal cardiac dullness is altogether effaced.

The character of the *breath-sounds* varies according to the nature of the opening; over the greater portion of the affected side the respiratory sounds are as a rule annulled, and in those cases in which the opening is small and quite valvular no auscultatory sounds may be detected at any point, although very often, even in these cases, at one spot a faint and distant hollow inspiratory sound may be heard on careful auscultation. In cases, however, in which the opening through the pleura is free, the entry and exit of the air to and from the pleural cavity gives rise to a variety of amphoric breathing, not loud, but peculiarly large and of metallic quality, which can rarely be mistaken. This amphoric breathing is most audible at some one portion of the chest nearest to the seat of perforation; it is commonly best heard at the mammary, or upper or lower scapular region, and is conducted more or less distinctly to a distance from this point. The expiratory portion of the amphoric sound is peculiarly indicative of a free and patent opening. In these latter cases, with free opening, the voice sounds may be attended with a metallic echo quite peculiar, whereas in the more valvular cases the voice sounds are not conducted at all. Metallic tinkling may sometimes be present and is a useful additional sign; a sign, however, which I have heard very typically yielded by large pulmonary cavities. Feeble, and more or less modified breath-sounds may be heard at the apex where there are still adhesions, and, immediately after the occurrence of perforation, friction sounds may sometimes be heard over portions of lung as yet in contact with the thoracic wall.

Displacement of the Heart—M. Gaide[1] was the first to describe displacement of the heart as an important sign of pneumo-thorax. It is indeed a constant and, save in exceptional cases in which the base of the opposite lung is consolidated, an essential sign of perforation of the pleura, and it is singular that it should have escaped the notice of such acute clinical observers as Laennec and Louis. Its occurrence simultaneously with that of the perforation, already noticed but not explained by M. Gaide, is a fact that would of itself cast suspicion upon the usual acceptance of the sign as being necessarily one of pressure. The cardiac displacement may be observed within a few minutes of the perforation, and is due in the first instance to the sudden removal from the mediastinum of the elastic traction of the lung which has collapsed, and the consequent unopposed traction upon it of the other lung. And, if the opposite lung be not solidified, the heart may from this cause alone be carried beyond the median line. Thus, I have recorded two cases, and have seen several others, in which the heart was displaced to the right of the sternum, yet in which, as proved by experiment *post mortem*, no intra-thoracic pressure existed.[2]

Progress.—If the patient survive the dyspnœa and shock of the first attack—and he usually does so, provided the opposite lung be not extensively diseased—after the lapse of some thirty-six hours or two days, signs of reaction fever, and hectic, announce the supervention of acute pleuritis and commencing empyema. The cause of the suppurative pleuritis which generally follows upon perforation of the lung is, partly the irritation of the air admitted into the pleura, and partly the tearing of adhesions, but the most potent cause is the escape of some of the contents of a cavity into the pleural sac. The signs of fluid in the pleura soon become apparent, and in some cases the pleura becomes rapidly filled with fluid, what air remains being compressed into such a small compass at the summit of the thorax as to be very difficult to detect. A large portion of the air in these cases is either absorbed, or as suggested by Dr. Duncan,[3] it may be expressed through the opening in the pleura. Dr. Duncan doubts whether the diseased pleura in pneumo-thorax can absorb air, but it must be remembered that such absorption is encouraged by the gradual pressure of accumulating fluid. Moreover, the aperture is frequently much too low down to permit of expression of any large portion of the air through it. Another effect of the pleurisy is, sometimes, to completely close the original perforation. The case may thus in the course of time be completely transformed from one of pneumo to one of pyo-thorax. In other instances very little effusion takes place and the signs of pneumo-thorax remain marked to the end.

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[1] "Observations de pneumo-thorax." Archives gén. de medicine, t. xvii., 1828.

[2] Medical·Times and Gazette, Aug. 21, 1869.

[3] Edin. Med. and Surg. Journ. vol. 28, 1827.

Diagnosis.—There can be but little difficulty in distinguishing a case of pneumo-thorax in which the effusion of air is extensive from any other disease. *Emphysema* is the only other disease in which we get hyper-resonance[1] and enfeebled breathing combined, but emphysema is a disease which affects both lungs; the respiration is never quite suppressed nor of amphoric quality, nor is the heart ever displaced as in pneumo-thorax. The diagnosis is sometimes difficult, indeed impossible, between localized pneumo-thorax and a large, thin-walled pulmonary *cavity*. Such cavities may yield almost tympanitic resonance, and very typical metallic tinkling rhonchus. Localized pneumo-thorax is most commonly situated, however, at the lower portion of the thorax, and in this situation such a large cavity as could be confounded with pneumo-thorax is of most rare occurrence, unless it be continuous from the apex downwards, in which case the heart's beat would be felt over an extended area on the *affected* side. The cases which I have most often seen mistaken for pneumo-thorax, however, have been cases of advanced phthisis, in which *acute congestion* has rapidly supervened at the base of the comparatively sound lung. Pain limits the movements and lessens the sounds over the newly affected part, there is considerable high-pitched resonance on percussion, and the symptoms may be precisely those of pneumo-thorax. Breath-sound and rhonchus can be heard, however, on careful auscultation, the heart is not displaced, nor is the percussion note truly tympanitic. Sometimes at first sight the dyspnœa of *asthma* nearly resembles that of pneumo-thorax, and, with general hyper-resonance, we may have in asthma an absence of respiration over portions of the chest; but the portions of lung so affected will vary in position, perhaps, even while we are listening, and the general wheezing râles present elsewhere, together with the history of the case and the effect of treatment, will prevent any real difficulty in diagnosis. I have seen more than one case of *hysterical dyspnœa* closely simulating pneumo-thorax, but the expression of countenance cannot be simulated, and a moderately careful physical examination will lead to a right diagnosis. The diagnosis of hydro or rather of pyo-pneumo-thorax from simple empyema is not difficult, the succussion splash, and the marked shifting of the dullness and resonance with change of position, being quite characteristic of the former disease. But, as already observed, some cases of apparently pure empyema have their origin in perforation of the lung.

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[1] The resonance of emphysema also lacks the drum-like note found in pneumo-thorax.

The diagnosis in pneumo-thorax, however, does not consist merely in separating it from other diseases, but also in distinguishing the kind of perforation that has taken place, and the probable existence, or not, of air-pressure within the thorax. The discovery of amphoric (to and fro) breathing renders it pretty certain that the opening is a free one, admitting the ready passage of air both ways, and that consequently no air-pressure is present. The complete absence of all breath-sound, with increasingly urgent dyspnœa, distended side and greatly displaced and oppressed heart, are equally significant of a valvular opening and of increasing intra-thoracic pressure. In a third set of cases hollow inspiration and obstructed rhonchoid expiration suggest a partially valvular opening.

Prognosis.—In the majority of instances, pneumo-thorax occurs towards the close of the disease, when the patient is already dying from extensive pulmonary lesions. Hence it is as a rule of speedily fatal augury. If in less advanced cases it occur on the side already most diseased, pneumo-thorax does not always shorten life. I have indeed seen several cases in which by arresting for a time the activity of the pulmonary lesions, its occurrence has considerably prolonged life. Of thirty-nine cases collected from the *post-mortem* records of the Brompton Hospital, the greatest duration of life was twelve months, four and a half, and four months in three cases respectively. The least duration ten minutes, fifteen minutes and six hours. The mean duration of the thirty-nine cases was about twenty-seven days.[1]

Cases in which a valvular opening is detected, are by no means necessarily the worst for prognosis, provided that they are promptly treated. The condition of the other lung is the point to ascertain in calculating the prognosis; if tolerably sound one may hope for arrest of the pulmonary disease and conversion of the case into one of chronic empyema.

A tendency to the accumulation of fluid may be generally observed, and it is possible that not a few of the cases of empyema that come before us in which after paracentesis we find evidence of apex disease, the empyema has originated in pneumo-thorax; the effect of the pneumo-thorax and subsequent effusion having been to arrest the pulmonary disease. I have met with two or three cases that would be best explained in this way:—In one instance, the patient left the Hospital "cured" after the removal of six pints of a sero-purulent effusion by paracentesis. He had presented, however, signs of early (or old?) disease at the apex of the lung soon after the tapping, and although he resumed his work for a time after leaving the Hospital, he again came under observation twelve months later with pneumo-thorax on that side, and died in three weeks.

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[1] On Consumption, etc. By R. D. Powell. Table of cases of Pneumo-thorax, p. 142.

Treatment.—Life is threatened on the occurrence of pneumo-thorax by *shock*, *asphyxia* and *exhaustion*, and these are special indications for treatment. The shock—which is due to the sudden lesion of a vital organ, and to the sudden dislocation and impediment to the action of the heart—must be treated by the administration of a stimulant, but above all things by an opiate. Opium is most valuable in calming the nervous system and in lessening the sense of dyspnœa. It is best given in the form of pill with camphor; in other cases divided doses of chlorodyne, or morphia subcutaneously may be administered. When the immediate shock has been relieved, the patient must be carefully examined and watched for signs of increasing pressure within the chest, to be treated by the timely introduction of a fine trochar. This trifling operation gives great, and curiously, sometimes lasting, relief. It can be repeated, however, if necessary, or a fine trochar guarded with gold beater's skin may be left in the side. If we bear in mind that the chief way in which positive pressure is brought about within the pleura, is by the thoracic wall on the affected side being expanded to the position of extreme inspiration and then recoiling upon the air pent up in the pleura, the advantage in these cases, when the excess of air has been removed, of strapping the affected side so as to control inspiratory movement, becomes obvious. A broad band of strapping firmly applied over the lower ribs, and reaching some two or three inches beyond the median line in front and behind, is sufficient to restrain the movement of the affected side.

As a rule pneumo-thorax occurs in persons already reduced in flesh and blood by previous illness; if the accident should occur at an earlier period of the disease, the venous engorgement, lividity, and general circulatory distress will be correspondingly marked. In such cases free dry cupping will give great relief. The portal system being the great reservoir for retarded blood, an occasional saline aperient is of value in pneumo-thorax, and also serves to correct the constipating effects of opium, the administration of which latter drug in repeated small doses, is on other grounds desirable for the first few days.

The intensity of the secondary pleurisy must be moderated by fomentations or poultices, and the associated fever, pain, and hectic, treated by occasional doses of quinine and opium, with salines or mineral acid as the case may require. The occurrence of effusion may be regarded rather with satisfaction than otherwise, provided the opposite lung be tolerably sound; some cases of one-sided phthisis have undoubtedly been arrested by the occurrence of pneumo-thorax and consequent empyema, the compression of the lung checking all active disease. Hence we should not be too hasty to remove the fluid by paracentesis, since, notwithstanding the presence of air, it has little tendency to become offensive, and the dyspnœa is lessened and the perforation more likely to become closed, by the substitution of steady for elastic compression of the lung. Paracentesis performed too soon, or with too great an anxiety to remove all the fluid, will lead to a recurrence of the pneumo-thorax by re-opening the old perforation. At a later period paracentesis may be necessary.

The frequent administration of food in small quantities with sufficient stimulant to steady but not to excite the heart's action, is needed to prevent increased suffering from exhaustion.

HÆMO-THORAX.

HÆMO-THORAX, or hemorrhage into the pleural cavity, may result from injury, or be secondary to certain forms of chest disease.

When traumatic in origin, the amount of blood poured out varies according to the vessel injured and the extent of the wound, be it heart, lung, aorta, intercostal or internal mammary artery. Such cases as these belong to the surgeon.

The diseases which may be complicated by hæmo-thorax are pleurisy (usually secondary to carcinoma or sarcoma), malignant disease of the œsophagus, pleuræ or thoracic parietes. It may arise from an apoplexy of the lung breaking through the pleura, or be due to the bursting of an aneurism. The quantity of blood poured out may be just sufficient to impart a ruddy tinge to the fluid effused in an ordinary pleurisy, or so abundant as to fill the pleura and compress the lung.

The symptoms of hæmo-thorax do not differ from those presented in other cases of pleuritic effusion, except the hemorrhage be profuse, when those indicative of internal hemorrhage are superadded. The physical signs are similarly those of fluid effusion into the pleura. The use of the fine needle syringe or aspirator is needed to make the diagnosis certain.

TREATMENT.—Observing the fluid of an effusion to be blood-stained, one would be unwilling to advise its removal except to an extent sufficient to relieve pressure symptoms.

CHAPTER VII.

BRONCHITIS—BRONCHIAL CATARRH.

ACUTE bronchitis is an active catarrhal inflammation of the mucous membrane of some portion of the bronchial tract

ETIOLOGY.—Bronchitis is a disease especially prevalent in Northern latitudes, in exposed and elevated situations, and in districts where moisture of atmosphere as well as a low temperature and cold winds prevail.

In our British climate these conditions are but too well fulfilled, and bronchitis is endemic amongst us. January, our coldest month, is that in which it is most prevalent. From the variable temperature and cold winds which characterize our spring weather, bronchitis is then also of common occurrence. In our autumn it is less prevalent, although persons with recurrent bronchitis get fresh attacks each year with the coming round of the cold weather.

The occurrence of bronchitis is influenced by *age, sex, occupation* and *condition of life,* chiefly in so far as these circumstances favor exposure to the known exciting causes of the disease, or render the individual less able to resist the action of such causes.

From the period of dentition onwards through childhood bronchitis is common. This is to be accounted for partly on grounds of lessened power of resistance, partly, as will be presently pointed out, in consequence of dentition itself, and in part also from the imperfect nasal development, and frequently obstructed nasal passages of children necessitating oral breathing. This abnormal method of breathing not infrequently remains a habit through life, and is a fertile source of chest troubles by the direct manner in which air is thus permitted to enter the bronchi and lungs, unwarmed by the nasal passages.

Old people have but feeble powers of resistance to the causes of bronchitis, and the disease is very prevalent and fatal amongst them.

The male sex is more exposed to the causes of bronchitis than the female, and suffers more accordingly.

Recurrent bronchial catarrh, associated with asthmatic symptoms, and with more or less emphysema, is well known to be an *hereditary* affection, and may occur very early in life. The winter bronchitis to which many individuals become subject at certain periods of life is also very markedly hereditary, and is no doubt really a phenomenon of senility. It cannot with these exceptions be said that bronchitis is hereditary. With regard to children my own observations would lead me to the opinion that the predisposition running through families is very often connected rather with the imperfect development of the nasal passages, already alluded to, sometimes also associated with enlarged tonsils, necessitating oral breathing, than due to any inherited delicacy of the bronchial membrane.

Cachexia of various kinds, gout, syphilis, phthisis, alcoholism, Bright's disease, must be ranked amongst the predisposing causes of bronchitis.

Of all the exciting causes of bronchitis, depression of temperature is the most important. The attack commonly supervenes upon exposure to sudden changes of temperature, or to wet, cold winds, especially in depressed conditions of the system, as after being over-heated by exertion, or exhausted by mental fatigue or shock. Dr. Sturges finds, from his careful analysis of the Registrar General's Returns for a series of years, that cold is a far more potent cause of bronchitis than of pneumonia. A large rainfall again, Dr. Sturges observes, increases the mortality from bronchitis, but does not affect the pneumonia rate. Bronchial catarrh is very prevalent in low-lying marshy districts, cold, wet weather with variable winds being most favorable to its occurrence.[1] "Catching cold" has been explained by Riegel in accordance with the experiments of Rosenthal, who found that the superficial vessels of animals exposed to a high temperature became dilated and partially paralyzed. Such animals on being removed to a cold room lose heat rapidly, their temperature becoming quickly reduced to below the normal by the rapid radiation of heat from the blood coursing through the surface vessels.

Whether the cold be "caught" in the head, or in the bronchi or lungs, or in some other internal organ is, in part at least, a matter of constitutional predisposition. As examples of the effects of local application of cold in determining renal congestion, I mav allude to the following cases:—

On the 20th of May, 1882, a youth aged twenty was admitted into Middlesex Hospital under my care with suppression of urine of thirty-six hours' duration, the suppression persisting up to the time of death on the evening of the 25th from uræmic coma. Post mortem, the kidneys presented the intensely injected blood-dripping appearance of acute nephritis. The boy had had scarlet fever when fourteen years of age, but there was no history of any renal complication. He had been for twenty-one months the subject of lymphomatous tumors involving the glands of the right side of the neck and pectoral region. For this intumescence more or less continuous cold had been applied by means of Leiter's tubes for five weeks, when the symptoms of suppression presented themselves. I had in my ward at this time another patient, a youth with exophthalmic goitre for whom I was employing the cold treatment, by means of the same useful apparatus. Frequent examinations of the urine were made, however, and after ten days slight albuminuria appeared, and the treatment was immediately suspended.

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[1] "The Natural History and Relations of Pneumonia," Octavius Sturges, M.D., F.R.C.P., 1876, p. 152.

The effect of prolonged chill to the feet in producing tonsillitis, and nasal or bronchial catarrh or pulmonary congestion, according to the natural predisposition of the subject, might be quoted as parallel examples with those related above. The influence of ice applications to the head, in reducing pyrexial temperatures, and the still more certain effects of the cold bath, may be likewise referred to in the same category, but on careful scrutiny of such effects of cold, whether under physiological or pathological conditions, one cannot feel satisfied with the mere blood-cooling hypothesis suggested by Rosenthal, but must invoke the more indirect action of the nervous system, to account for the phenomena.

Dusty employments lead to chronic catarrh of the proximal air passages, and if long continued, such catarrh may extend more deeply into the lungs. It is very instructive to note, however, how out of those who are exposed to obviously mechanical influences of this kind, many escape unharmed. In noting the history of patients who have been suffering from irritative bronchitis, I have found instances in which the fathers of the patients had passed their lives at the same occupation without complaint. Hirt has observed that the inhalation of injurious gases, and even of dust, may after a time fail to excite catarrh, the workmen becoming as it were acclimatized to such conditions.[1] The second or third generation seems to become more vulnerable to such given influences.

Many a fresh catarrh, in cases of confirmed pulmonary disease, may be traced to dusty winds, or to the irritating fogs which occasionally prevail in this metropolis. Blood inhaled to distant bronchial tubes during an attack of hæmoptysis, or from hemorrhage during tracheotomy, may set up bronchitis. A very virulent bronchitis, attended with profuse expectoration, is occasioned by the passage of acrid septic matters from fœtid pulmonary or pleural cavities over the bronchial surfaces.

Acute bronchial catarrh, commonly supervenes upon, or forms a part of, the epidemic disease influenza. Perhaps in this category hay asthma should be included as a specific catarrh, due more distinctly than influenza to the reception of a particulate poison. Many of the specific fevers, measles, whooping cough, enteric fever, small-pox, are attended usually in the early or eruptive periods with bronchial catarrh; the bronchial mucous membrane in measles and small-pox, being often involved in the exanthematous eruption. During the first dentition some children have repeated attacks of bronchial catarrh, coincident with the eruption of each tooth; just as other children suffer at these times from nasal catarrh, or others again from intestinal catarrh. It may be urged that, during this period, children are frequently the subjects of irregular febrile disturbance which renders them more liable to chills, and that this period is usually chosen too for the change from long clothes to naked legs and chest; but for the most part the catarrhal affections prevalent at this age cannot be explained otherwise than as being of reflex nervous origin, and the fact that under the same circumstances we may get convulsions in the place of either of the above occurrences, is strongly confirmatory of the truth of this view.

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[1] "Krankheiten der Arbeiter," Breslau, 1873, quoted at length by Riegel in Ziemssen's Cyclopœdia, vol. iv., p. 312.

Certain heart diseases, mitral regurgitation, and in a still greater degree mitral stenosis (a disease, I am persuaded, often congenital,) predispose to bronchial catarrh by obstructing the return of blood from the lungs, thus causing mechanical congestion of these organs and of the small bronchi. Finally, it is very important to remember that one attack of bronchitis predisposes to future attacks.

The morbid appearances of acute catarrh are but rarely to be observed *post mortem*, but they may sometimes be seen in perfection in the trachea by means of the laryngoscope during life.

(1) In the first stage there is hyperæmia of the mucous membrane with œdema of the basement layer. Upon hyperæmic swelling of the mucous follicles in part depends the temporary check to secretion, and probably the ducts of the follicles are also for a time occluded by the swollen condition of the membrane they traverse. In this stage the affected membrane is minutely injected, swollen, and unduly lacerable, presenting in some severe cases, especially in young children, minute hemorrhages.

(2) The mucous flow is soon increased, however, and is mingled with sanious exudation from the basement layer, and sheddings of columnar epithelium, and of mucous cells derived from multiplication of the deeper cells of the epithelial layer. Thus is constituted the muco-purulent "secretion," of bronchitis, thin and glairy at first, thicker and more opaque after a few days. To the naked eye the appearance of the mucous membrane is more slaty in hue, covered with secretion, sanious or thick, according to the stages referred to.

(3) The tissues of the bronchial tubes beneath the basement membrane do not remain passive whilst these active processes are going on in the layers above them. The connective tissue cells multiply, and the endothelium of the lymphatic spaces of inner and outer fibrous layer proliferates; the products of such proliferation do not, however, find their way to the surface, being unable to penetrate the basement membrane, but collect in the interstices of the cellular tissue, and fill up the lymph

paths along which they slowly course towards the sub-pleural lymphatics.[1]

By these processes, thickening of the bronchial membrane is effected, together with that thickening of lung texture spreading from the bronchial sheaths, which forms a part of the pathology of hypertrophous emphysema.

Acute bronchitis may be conveniently divided according to Walshe into:—

(*a*) Bronchitis of the larger and medium sized tubes.

(*b*) General and capillary bronchitis.

To these should be added—

(*c*) The acute asthenic bronchitis of the young and aged.

a. The *symptoms* of acute catarrh affecting the large bronchial tubes are commonly, in the first instance, those of an ordinary cold in the head; chills of a creeping character, never amounting to a rigor, occur from time to time on the first day, and are attended with a feeling of malaise, a somewhat hurried pulse, slight soreness of the throat, sneezing, and coryza. The temperature is raised a degree or so above the normal, but although there is thirst, aching, perhaps pains in the limbs, and a considerable sense of feverishness, the febrile phenomena are really very slight in adults, more decided in young children. After some twenty-four or forty-eight hours the patient complains of a soreness, or rawness as it is usually more accurately described, felt behind the upper sternum, accompanied by a sense of constriction or oppression in this region. The cough is frequent, dry, and is attended with more or less pain of a rending character. The voice is deepened and sometimes husky or suppressed, and the breathing is perceptibly quickened. On the second or third day secretion takes place, and with the expectoration of a thin aërated mucus the patient soon experiences a marked sense of relief. In fact the pyrexial stage has already passed, the pulse is quiet, the cough loose, and expectoration easy, the mucus expelled becoming more opaque and semi-purulent. The secretion subsides, and the cough gradually lessens, and usually disappears in a week or ten days, but in the morning, after his first sleep, the patient still feels some oppression in the chest, and is not relieved until he has brought up some purulent mucus.

As regards physical signs we may hear a few dry, sonorous rhonchi vibrating through the chest, obviously produced in the larger tubes; and if only the largest bronchi be affected, no râles of any kind are heard. When the secretion is more established the rhonchi are looser, modified or for the moment removed by cough, and accompanied, especially over the base of the lungs, by scattered, muffled, bubbling râles. The percussion over the chest is unaltered, and the râles are symmetrically distributed over the two sides, but more abundant over the bases of the lungs.

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[1] Hamilton. "Pathology of Bronchitis," p. 33, et. seq. Dr. Hamilton goes very minutely into this important part of the pathology of bronchitis.

b. When the acute catarrh affects the *smaller and capillary bronchial tubes* the dyspnœa becomes marked, with lividity of lips and an anxious, distressed expression of face, the nares expanding with each respiration. In feeble and old people, there may be considerable systemic shock with general prostration, reduced temperature, and rapid, feeble pulse. In more robust persons the febrile action is at first decided, although the temperature rarely mounts above 101° or 102°; the pulse is full and not very quick, between 80 and 100 in the minute, the respirations being rapid out of proportion to the pulse. The cough is frequent, and is very soon accompanied by the expectoration of a viscid adhesive mucus, difficult to get rid of. The digestive functions are impaired, the tongue furred, the bowels usually confined, and the urine loaded with lithates.

On inspecting the chest, the thoracic movements are observed to be increased both in frequency and depth, the diaphragm partaking but little in the respiratory efforts. The explanation of this is soon found, for it is with the front and upper parts of the lungs that the patient is principally breathing. The resonance on percussion is everywhere unimpaired, it may be even increased. Fine bubbling râles are audible over both posterior bases: to a much less extent, or not at all over the upper and anterior portion of the chest, where, however, sibilant and sonorous rhonchi prevail. This distribution of the râles in bronchitis is mainly a question of gravitation. Dr. Walshe has well observed that even in bronchitis of mild type and not involving the capillary tubes, we may still get fine bubbling râles at the posterior bases from gravitation of the secretion to the minute tubes.

The *diagnosis* of bronchitis of this degree depends upon the symmetrical distribution of fine bubbling râles not associated with any percussion dullness, or bronchial quality of breath-sound, and with but a moderate rise of temperature.

The diseases which may be confounded with it, are pulmonary œdema, tuberculosis and acute phthisis. If œdema be limited to the lungs, however, it must be dependent upon cardiac defect, the history and signs of which must be looked for. Tuberculosis is the disease most readily to be mistaken for bronchitis, for the *signs* of acute pulmonary tuberculosis are almost identical with those of capillary bronchitis. If the tuberculosis of the lungs be only a part of a general distribution of tubercle, the special adynamic symptoms of that disease will be apparent. When restricted, or mainly so, to the lungs, the excessive dyspnœa and great prostration occurring in a patient in early adult or middle life are suspicious signs. The temperature may not help us at all, for in some cases of pulmonary tuberculosis it is not high. But, if present, a high temperature, especially when accompanied by profuse sweatings, always means something more than simple bronchitis. The distribution of physical signs is, however, in tuberculosis somewhat different, and their characters are not quite the same. With urgent dyspnœa we may have comparatively few moist sounds, and these are quite as marked at the apex as at the base, and sometimes more so. There may be at one apex some evidence of previous pulmonary disease. With only scattered sonorous or sibilant râles over the chest, or these accompanied with a short inspiratory crepitus, we have, in tuberculosis, quite a disproportionate amount of dyspnœa, with marked recession of soft parts. The history of attack and other features of the case, however, exclude asthma.

Acute phthisis cannot, after attentive auscultation and percussion, well be confounded with bronchitis. More or less pneumonic crepitation, with patchy tubular breathing and larger clicks, are superadded to the catarrhal bronchial râles, and the signs are distinctly more advanced at some one portion of the chest, whether this be the apex or base. The fever again is characteristically high and remittent.

c. Acute asthenic bronchitis (suffocative catarrh) is a malady commonly met with at both extremes of life, and in the aged, especially, it is one of the most fatal of diseases. A fairly typical example may be thus related in illustration of the salient features of the disease.

Mrs. M., a widow, aged 76, of thin, spare build and of previous good health, was dining out with friends on the evening of December 27th, feeling in her usual good health and spirits. She had never before suffered from any chest illness, but, it was believed, that on her way home she became chilled. Mrs. M. appeared, however, well the following morning, but towards the latter part of the day (28th) felt drowsy and somewhat chilly; she became more obviously ill in the course of the evening, the breathing being quickened and oppressed, and towards midnight her symptoms became so aggravated that the doctor was sent for. Mrs. M. was now found to be in a state of profound collapse with small, feeble pulse, cold extremities, low temperature and sweating surface. The respirations were quick and shallow, the countenance anxious, with some lividity of mucous membranes. No morbid sounds save a few wheezing rhonchi were to be heard over the chest.

A free recourse to brandy and other stimulating remedies rallied the patient so notably, that the friends became hopeful, and the doctor plied his remedies and planned out food, physic and stimulants with a cheery exactness. By mid-day or evening the chest signs had become more marked, short inspirations being followed by prolonged wheezing expirations, the pulse keeping steady at about 90 beats a minute. The cough and expectoration were now troublesome and difficult, preventing, as the night advanced, more than brief snatches of sleep. The body temperature rose to about 100°, and it was observed that the patient wandered in slight delirium, from which, however, she could readily rouse herself. Fine, bubbling râles were now, at the close of the second or third day, audible over the chest, most abundantly and most definedly at the posterior bases, where but very little air could be heard to penetrate. The respiratory movements were peculiar. The chest lifted quickly by the action of the superior auxillary muscles, whilst the bases of the thorax receded. The long wheezing expiration which followed was apparently produced by the downward recoil of the chest upon the diaphragm pushed upwards to meet it by the contraction of the abdominal muscles.

Tracheal rattles soon became developed, removable at first temporarily by cough, but soon to return. The pulse quickened, the patient became more exhausted, with muttering delirium or incoherence, and death closed the scene at about the 70th hour.

Such is a sketch of the phenomena presented by a typical case of this fatal malady, the characteristic features being, (1) the first shock of the disease, causing almost fatal collapse at about the twelfth hour of attack; (2) the rallying of the patient, and the oncoming of the signs of general bronchitis, attended with slight febrile reaction; (3) at about the third day the appearance of signs (labored ineffectual thoracico-abdominal movements, with filling of lower bronchi) of bronchial paralysis and of the circulation of venous blood through the nerve-centres. No doubt this later circumstance accounts for the forcible respiratory efforts, far beyond the strength of the patient, that come on at this third fatal period, and continue to the very end. It is rare, indeed, for recovery to ensue in any case in which this character of breathing has once been observed.

Young children not unfrequently succumb to the first shock of bronchitis, being overwhelmed before their illness seriously attracts the attention of their parents. A considerable proportion of the infants brought to our hospitals dead, or in a dying condition from sudden illness, are found *post mortem* to present no other lesions beyond the signs, very slightly marked, of early bronchitis. A few petechial spots in the mediastinum, pericardium or pleura, may testify to the brief struggle of the little patients.

The *prognosis* in bronchitis of whatever degree, is, in adults, generally speaking, favorable. Capillary bronchitis in very young and in old people is, on the other hand, very frequently fatal, yet there is perhaps no disease whose mortality is more influenced by treatment than that we are now speaking of.

Taking the capillary bronchitis in the adult as our type in regard to *treatment*, the first thing to be seen to is the warmth of the room; a fire and a steam-kettle are the first things to procure, so as to raise the temperature of the room to between 65° and 67° F., and immense relief is given to the patient by this means alone. Due care must be taken, however, to ensure a proper supply of air as well as to preserve a uniform temperature. The use of the steam-kettle is not only to moisten the air of the room, but in most bed-rooms it is the only possible means of raising and maintaining the temperature at the desired height. A large mustard or mustard and linseed poultice should be applied to the front of the chest, or to the back, and followed up by hot linseed applications or cotton-wool to the chest. In children a jacket poultice is often very useful, but one must not forget that both in young or weakly children, and in old people, a linseed poultice wrapped round the chest may be a very serious impediment to free thoracic movements, and in such cases it is often more judicious to have recourse to cotton-wool covered with oil silk and an occasional mustard, or mustard and linseed poultice, to keep the blood determined to the surface.

As regards drugs, a saline mixture with ipecacuanha is the best to begin with. In strong adults antimony wine is very useful, especially in the dry stage of the catarrh, and should be given in small doses at frequent intervals. In old people, on the other hand, we must usually add ammonia to our prescription. The special danger in infants arises from the possible occurrence of pulmonary collapse and secondary broncho-pneumonia. The collapse arises from want of power of expectorating, and the resulting broncho-pneumonia is just as mechanical in its origin, being dependent upon the determination of blood, during the thoracic efforts at expansion, to those parts of the lung which cannot expand on account of their bronchi being plugged. The timely administration of ipecacuanha emetics, if the secretion be abundant, will avert this danger. Friction, with stimulating liniments, such as the ammonia or acetic turpentine liniments of the Pharmacopœia, if necessary further diluted, is of great service in young children after the first stage has passed.

In old people danger arises principally from exhaustion or paralysis of the bronchial tubes, the latter, perhaps, being but another evidence of exhaustion. To avert these dangers, we must, from the first, support the patient by the frequent administration of nutritious liquids, and by the timely employment of stimulants in carefully regulated doses. Of all alcoholic stimlants, brandy is certainly the best for this purpose. The administration of opiate remedies in bronchitis should, as a rule, be avoided, and absolutely so in cases in which lividity of lips shows already defective aëration of blood. In young children and old people opium must be used for bronchitis with the utmost caution. As a sedative at night, bromide of ammonia with aromatic spirits of ammonia is one of the best we can choose. Chloral is not very suitable in acute cases, but a small dose combined with bromide of ammonia will suffice to give rest without risk of depressing the heart's action too much. When the heart fails, and symptoms of over-loading of the right ventricle present themselves, digitalis may be usefully given, stimulants persevered with, and dry cupping may be tried with advantage. In a few cases, I have certainly seen good results from the substitution of belladonna for digitalis. In certain cases, in which there is marked venous plethora, with a weak and failing heart's action, from an over-burdened right ventricle, venesection is attended with manifest relief.

Next to avoiding a fatal issue, our efforts should be directed to preventing the case going on to chronic bronchitis, especially in those who have had previous attacks. When the acute stage is past, some patients at once convalesce without any special treatment. In other cases the secretion continues abundant and more purulent. The saline mixture must now be given less frequently, or changed for a more stimulating expectorant containing senega and ammonia, and some mineral acid with calumba or quinine, ordered to be taken twice a day. The turpentine acetic liniment is of great value in this stage, its usefulness being partly, I suspect, due to inhalation of turpentine vapor.

In chronic cases, with profuse muco-purulent expectoration, much advantage is gained by the use of tar, which may be taken in the form of pills (*British Pharmacopœia*) or in Guyot's or Jozeau's capsules. The resinous preparations are also sometimes useful in this stage. In many cases it is best, however, to leave the bronchial mucous membrane alone, and to direct treatment towards improving digestion and appetite.

In all cases of secondary bronchitis we must direct our treatment with due regard to the removal or amelioration of the more general disease which underlies the bronchial affection. Cases of secondary bronchitis are, as a rule, chronic, with acute exacerbations.

With bronchitis secondary to gout or kidney disease, to alcoholism, to heart disease, and to already existing diseases of the lungs, I will not further deal. The affection of the bronchial mucous membrane is, in such cases, most commonly a part of a much wider affection. It is the primary disease that we should especially treat, and to direct too much attention to the pulmonary condition would apparently sanction an erroneous view of such cases.

Dust Bronchitis.—In certain cases of exposure to dusty employments, the whole effect of the irritant falls upon the bronchi. Habitual cough with recurring bronchitis and subsequent emphysema, paroxysms of asthma, etc., are the leading phenomena in these cases, and are commonly met with amongst potters, miners, flax-dressers, millers, plasterers, masons and others.

The following case is not an unusual one of bronchitis and asthma induced by the inhalation of dust.

William C——, aged 38, residing in London, admitted into the Brompton Hospital under my care January 23rd, 1877. Patient was a man of temperate habits, married, one child living: his wife had had one miscarriage, but there was no history of syphilis. He had been for twenty years employed as a fret-cutter. Four years previous to admission, he had had an attack of bronchitis, and since that time he had suffered constantly from cough, with suffocative attacks nearly every night.

Patient had never had hæmoptysis but had lost flesh considerably. His father died of rheumatic gout, and his mother of dropsy. There was no phthisis in the family. On admission, patient complained of cough, which became worse and was attended with severe attacks of dyspnœa at night. Expectoration moderate. Occasional night-sweats. Appetite poor, bowels constipated.

He was a tall and fairly well-built man, with a somewhat suffused countenance and breathless look. The chest was well formed, but expansion with inspiration impaired. Right semi-circumference, an inch and a half above the nipple level, 15¼ inches, left 15¾ inches. Right ditto, two inches below nipple level, 16 inches, left 16¼ inches. Percussion note over the front of the chest was hyper-resonant, the resonance extending on the right side a hand's breadth below the nipple, on the left side, over the normal area of heart's dullness, and inferiorly to the costal margin. Diffused sibilant râles were audible front and back, and at the posterior bases some mucous râles were heard. At the time of my visit on the 26th of January, the patient had been in Hospital three days, and was suffering, as before admission, from nightly attacks of dyspnœa, and from troublesome cough. He was on the ordinary full diet of the Hospital. The *Mistura Potassii Iodidi cum Stramonio* of the Hospital Pharmacopœia, containing three grains of iodide of potassium, ¼ gr. of extract of stramonium to each dose, was now ordered to be taken at 12 noon, 4 p. m., 8 p.m., and 12 midnight, the diet remaining the same, and from the next night he had no serious dyspnœa. On January 31st, a note is entered. "Patient feels much better, and dyspnœa almost gone, cough easier." February 3rd, "has not felt suffocating sensation for the last six nights." He continued the mixture, however, for a month, and then took it in half doses for another fortnight. On February 8th, respirations were free and unaccompanied by râle, and by the end of the month the cough had disappeared.

March 1st. Right semi-circumference above nipple, 15⅛ in., left, 15½. Right semi-circumference below nipple 16⅓, left 16⅓.

Expansion above nipple level, ordinary inspiration ⅛ inch; deep inspiration 1⅛ to 1½ inch. Below nipple level no movements on ordinary inspiration, one inch expansion on deep inspiration. Weight on admission, 8st. 9lbs.; on leaving Hospital, 9st. 2 lbs.

Unfortunately I did not, in this case, *measure* the mobility of the chest on admission. But my observation of "expansion with inspiration impaired" refers to total mobility of chest, and the circumferential measurements at that date may be taken as those of a chest fixed by the emphysematous condition of the lungs. The later measurements, however, which were similarly taken in the position of repose are almost identically the same; but at this period the mobility of the chest, its capability not only of expansion but *also of contraction*, was nearly equal to that of health, and bore valuable testimony to the recovery of the lungs in vital capacity, a point not to be estimated by reference to chest expansion alone.

Again, it may be observed in this case as in many others, that mere rest from his dusty occupation did not suffice for the patient's recovery; although doubtless had he remained in pure air a sufficient length of time, he might have recovered without treatment. His attacks had the paroxysmal character peculiar to asthma, coming on towards the small hours of the morning, *i.e.*, after a certain period of repose, whilst the breathlessness and cough of the bronchitis and attendant emphysema were constant through the day. The effect of the stramonium and iodide of potassium mixture was very striking and immediate. Having regard to the period at which the dyspnœa became distressing, the mixture was so ordered that by midnight he had taken in the course of twelve hours a grain of stramonium extract, and twelve grains of the iodide. To which of these two drugs, both active in the relief of asthma, the amelioration of symptoms was due I am unable confidently to say. The mixture was not prescribed experimentally, but as a well-tried and trusted remedy in similar cases.

The mechanism of the dyspnœa, however, was pretty obvious; the man had an unduly secreting, and probably a somewhat thickened, bronchial tract, with great irritability of the bronchial muscular apparatus, and constant tendency to spasm of the tubes. At a certain time after repose, secretion would accumulate and give rise to spasm. The hypersensitiveness upon which the bronchial spasm depended was at once lessened by the stramonium, whilst the iodide had a more permanently alterative action upon the mucous membrane and its secretion. This explanation would be perhaps generally accepted, but abundant experience with regard to iodide of potassium assures me that it alone frequently suffices very rapidly to relieve asthma. In what way the drug does this is, so far as I know, entirely unexplained. The patient left the Hospital well, but after a time, failing to find other work, was reduced to the necessity of recommencing his old employment, and soon had a return of all the old symptoms for which he was treated elsewhere by ordinary remedies (ether and expectorants) without avail.

In the trade of fret-cutting the operator has constantly, with his mouth, to blow away the fine wood dust that collects upon his work, and thus necessarily inhales much of the dust. The patient above referred to, stated that he could distinguish by the taste the different kinds of wood, and he found rosewood (the taste of which he compared to cayenne pepper) to be the most irritating. Walnut wood was more astringent and bitter, but less irritating. I have since seen this patient in fairly good health, having for some time abandoned his former work. There does not seem to be any reason, however, why a respirator perforated with a tube, should not be so adapted as to enable such patients to carry on their occupations without injury to health.

PLASTIC BRONCHITIS.

Apart from croup and diphtheritic conditions, plastic exudation into the bronchial tubes is of rare occurrence. It is said to be more common in females than in males.

It may occur at any age from infancy to advanced life, and is not necessarily associated with any obvious derangement of the general health.

PATHOLOGY.—The morbid lesions resulting in the production of fibrinous casts of the bronchial tubes are:—1. Inflammation of the bronchial membrane in hyperinotic subjects. 2. Hemorrhage into the bronchial tubes. The casts are found in the smaller and medium, rarely in the larger, tubes, and never extend to the trachea. They are fibrinous in character, usually laminated, solid or tubular, and often speckled with blood.

SYMPTOMS.—Troublesome straining cough coming on in paroxysms, with great dyspnœa, also of a paroxysmal character, and the expectoration of gelatino-fibrinous looking masses which, on floating out in water, are found to present moulds of the bronchial tubes, constitute the essential symptoms of plastic bronchitis; and, until the latter phenomena of actual expectoration of casts is presented, there is nothing that can be said to be truly characteristic, in the symptoms of this malady. The bronchial casts are expectorated frequently in small pieces, with the interposition occasionally of larger and more characteristic moulds. The smaller fragments clothed in mucus may readily escape observation (Walshe). Hæmoptysis in streaks may occur, or a decided hæmoptysis may precede the appearance of the casts.

A noticeable feature of the disease, in most cases, is its chronicity, or, rather, the long period, weeks, months, or even years, during which the patient may suffer recurrences of the plastic attacks; each attack being attended with the symptoms above described. A mild degree of pyrexia may be associated with each attack.

PHYSICAL SIGNS.—The signs of this malady are very indefinite, being those of bronchitis affecting one base, or of broncho-pneumonia. In some cases a certain degree of dullness, with very feeble respiration, may be present. It cannot be said that there are any physical signs proper to this malady.

PROGNOSIS.—As a rule these patients recover, but the disease may ultimately terminate in phthisis, or any particular attack may proceed to broncho-pneumonia.

TREATMENT.—The treatment of this disease is that of bronchitis, salines and alkalies being indicated, and hot moist inhalations calculated to give relief.

CHAPTER VIII.

ON PULMONARY VESICULAR EMPHYSEMA.

PULMONARY vesicular emphysema may be defined as a dilatation of the air-cells of the lungs with antecedent or associated atrophic changes, or, more briefly still, as a dilatation of the lung proper with textural atrophy.

This disease may in its acute form affect persons of any age; in its chronic and more permanent form it affects, most commonly, persons in middle or advanced middle life. Atrophous or senile emphysema, which scarcely merits consideration as a variety of the disease, affects only aged people, and is indeed but a part of the senile state.

PATHOLOGY.—The perfectness of the function of respiration consists quite as much in the power of contracting as in that of filling the chest, and it is this power of contracting the chest that is lost in emphysema. The lungs having lost their reserve elasticity, no longer tend further to contract at the completion of expiration: nay, expiration is never completed, the thoracic parietes and diaphragm instead of being drawn inwards by the traction of the lungs recoil simply to their position of repose, and oppose their dead weight to the inspiratory muscles instead of aiding the action of these muscles by their elastic rebound. Hence in extreme emphysema the inspiratory act, commencing at the point where in health calm inspiration would end, has to overcome, (1) what remains of the elastic resistance of the lungs, (2) the inertia of the parietes, and (3) the elastic resistance of the parietes: instead of, as in health, having to deal with the elastic resistance of the lungs alone, and being in this work aided by the outward spring of the ribs. (See p. 6). Consequently, in marked cases the breathing is always forced and more or less difficult. Let me now briefly recount the conditions present in emphysema, making such additional comments as seem called for.

(1) The lungs in all cases of emphysema are permanently expanded to about the position of ordinary inspiration, their elasticity being, so to speak, relaxed to this point. The individual air-cells are correspondingly enlarged and the pulmonary vascular system lengthened.

The enlargement of the air-cells commences, as pointed out by Rindfleisch,[1] in the central infundibular cavities which, normally about one-third larger than the alveoli opening into them, become proportionately much larger. The alveoli themselves next enlarge, their septa become withered, and the lung conformation is thus simplified.

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[1] Pathological Histology, vol. ii., p. 7. (New Syd. Soc. Edition).

(2) The texture of the lung is impaired more or less.

In acute emphysema loss of tone from repeated over-distension, *e.g.*, during paroxysms of coughing, is the chief defect present, and, in young persons especially, is speedily recovered from.

In cases of long-standing emphysema, resulting from repeated attacks of asthma or bronchitis, what may be regarded as the second stage of the disease is entered upon; the nutrition of the lung suffers, many of the small vessels become obliterated, withering backwards from their capillaries, the alveoli atrophy, the partitions between adjacent infundibula or alveolar spaces become thinned and finally give way, and the spaces here and there coalesce to form blebs or small cavitations, with thread-like remnants of vessels crossing them, resembling in miniature the trabeculæ of tubercular cavities. Where the disease has resulted from oft recurrent catarrhs, commonly with a history of a distinct attack of bronchitis at the commencement, ill-developed fibrous tissue, the result of repeated and long-continued congestion, toughens the lung texture, assists in impairing elasticity, and partakes in the subsequent degeneration.

In cases of constitutional or hereditary origin, the atrophic changes originating in fatty degeneration of the epithelium and vessels of the lungs, precede or accompany the catarrhal phenomena. This impairment of the texture of the lung renders any dilatation, however induced, more or less permanent. In these cases of primarily impaired lung texture, enlargement of the chest to the limits of thoracic resilience takes place quite insidiously, and the chest thus assumes permanently, by imperceptible degrees, the position of moderate inspiration. It must not be forgotten, too, that the ribs themselves, and especially their cartilages, often prematurely undergo textural changes of a degenerative kind, the result of which is an increased rigidity and a straightening of the rib arch, and thus an enlargement of the thorax.

The chief results of the above pathological processes are loss of elasticity of lung and obliteration of vessels, the latter result being in part due to primary atrophic changes in the capillaries extending back to larger vessels, in part to secondary atrophic changes from narrowing of the blood-current as the vessels become lengthened, thus causing them to wither from deprivation of proper blood-supply.

(3) In consequence of the relaxed elasticity of the enlarged lungs they no longer exercise any traction upon the mediastinum except during inspiration. Hence an important, because constant, aid to the return of blood to the heart is lost.

Many authors go further than this, and hold that the large lungs in emphysema are, so to speak, pent up in the chest, and exercise pressure upon the heart between them, and upon the ribs and diaphragm which enclose them. A little reflection and clinical observation will, however, render it clear to anyone that this supposed pressure of the lungs upon the surrounding parts is, if not impossible, of infinitely rare occurrence and minute degree. The enlarged thorax, flattened diaphragm, and lowered heart, are all phenomena due to defective recoil of the lungs, not to their forcible distension: and are to be observed in health on the lungs being inflated during deep inspiration, which position is retained in emphysema.

(4) In addition to the negative impediment to the circulation referred to in the preceding proposition, there is a positive impediment in the stretched, and in part obliterated, capillaries of the lungs.

(5) In consequence of the two last-mentioned conditions the venous system is over-full and the over-worked right heart thickens. The increased power of right ventricle at length becomes, however, inadequate to contend against the ever present, and from time to time (during bronchitis or asthma) increased, resistance to the pulmonary circulation. The whole venous system becomes engorged, especially the hepatic and portal systems; the circulation is carried on at a heightened pressure, venous hemorrhages occur, and œdema, commencing at the legs, sooner or later sets in.

(6) Another effect of the relaxed state of the lung as regards elasticity in emphysema, is to interfere with that condition of permanent patency, in which the small bronchi are normally held by the constant traction upon them of the elastic lung from all sides. In emphysema this traction becomes, in expiration at first, entirely neutralized, and in marked cases collapse of the bronchioles must occur during expiration, thus accounting for the labored and obstructed character of that portion of the respiratory act.

(7) *Compensatory changes in emphysema.*—Amongst the consequences of emphysema those which are of the nature of compensation must not be forgotten. So ready are the adaptations of our economy to altered circumstances, that it may be said that emphysema alone does not kill. (*a*) As the capillaries become partially obliterated, fresh communications are opened up between the neighboring vessels corresponding to them, by the formation of fresh anastomotic loops, or by the widening of communications already existing. The pulmonary and systemic veins communicate normally on the walls of the smaller bronchi. With the formation of pleuritic adhesions, so common in this disease, new loops of vessels connect the pulmonary with the systemic circulation, and along the insertion of the diaphragm especially, and margins of the sternum, fringes of enlarged vessels mark the connection between the two circulations. Thus, in some measure the pressure of the pulmonary circulation is eased. (*b*) The hypertrophy of the right ventricle of the heart above alluded to, is up to a certain point of a strictly compensatory nature, serving to over-

come by increased power the increasing impediment to the pulmonary circulation. Thus, for a time at least, equilibrium is maintained, to be disbalanced again and again, however, in most cases by intercurrent attacks of bronchitis or other cause of over-strain, until at last the limits of compensatory recuperation are passed.

ETIOLOGY.—When we come to inquire how this over-expansion of the air-vesicles is produced, we are met by various explanations, none of which alone is sufficient to explain all cases. Sir William Jenner has shown that *expiratory effort*—during straining or coughing, particularly the latter—is an efficient cause of general emphysema, those portions of lung which are least supported, viz., the apices and anterior margins, and also the parts corresponding with the comparatively yielding intercostal spaces, becoming first affected. But with the production of emphysema in these portions of the lungs a shifting of the relationship between the lung and the thoracic surface takes place, and parts which were originally in apposition with the ribs come to be opposed to interspaces, and in their turn yield before the distending force of air pent up and compressed during cough.[1] Again, in the production of local emphysema, *inspiratory pressure* is undoubtedly an important agent. Thus, if one lung be disabled or bound down by some inflammatory process (as old pleurisy or chronic pneumonia) the other lung perforce becomes more capacious; whether this extra capacity shall be derived from true hypertrophy or mere dilatation (emphysema) depends upon the nutritive vigor of the patient. During bronchitis certain of the air-tubes may become occluded by mucus, and the inspiratory force then operates as a distending power upon the remaining portions of the lung until the deficiency in air-space is compensated for. Professor Rindfleisch, who adopts Dr. Gairdner's inspiratory theory in regard to the production of emphysema, further holds that "during the antecedent bronchitis, first one, then another bronchial tube is plugged with secretion, and so first one, then another segment of the lung is subjected to an abnormal degree of (inspiratory) distension."[2]

In the histories of the majority of cases of pulmonary emphysema, however, facts are wanting which would justify our accepting the *inspiratory* cause as effective in their production. When secondary to repeated attacks of bronchitis, the tussive expiration is a much more powerful cause of over-distension of lung than any inspiratory efforts (short of that prolonged and urgent dyspnœa, which is not met with in these cases) could be. Moreover, both the expiratory and inspiratory theories imply the presence of some pre-existing bronchitis, or some local lesion disabling a portion of the lung, or the pursuance of some occupations requiring repeated efforts, during which the glottis is closed and the chest compressed, all of which are wanting in a considerable proportion of cases.

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[1] Sir William Jenner (Reynold's Syst. of Med., vol. iii., p. 478) refers to Mendelssohn as having in a paper, Der Mechanismus der Respiration und Circulation, in 1845, anticipated him in this view respecting emphysema. The authorship and advocacy of the view in this country rests, however, with Sir W. Jenner.

[2] Manual of Pathological Histology, vol. ii., p. 6. (New Syd. Soc. Edit.)

In an interesting paper read before the Belfast meeting of the British Medical Association, Dr. McVail of Glasgow also advocates the inspiratory theory of emphysema. Brit. Med. Jour., Nov. 15, 1884.

In truth, the emphysema frequently precedes the bronchial affection, although it is subsequently aggravated by the first attack of bronchitis; and, admitting with Drs. Jackson,[1] Greenhow,[2] Waters[3] and others, that a *failure of nutrition* is in a large proportion of cases the predisposing cause of emphysema, we need go but little further to explain the occurrence of that disease in its earlier grades. The effect of damaged textural nutrition of the lungs is to relax them by impairing their elasticity, and, as I have elsewhere shown, the thoracic elasticity or resilience tends to expand the chest to the degree of from one to four millimeters in each direction. As the lungs relax their elasticity, they yield to this traction exercised upon them by the thoracic wall, and to the weight of the abdominal organs dragging upon the diaphragm, and thus we get expansion of lung to such a degree that any extension will suffice to cause symptoms of dyspnœa. Impairment of a degenerative kind in the elasticity of the ribs and cartilages, referred to by Freund, may take place *pari passu* with, or may even precede, this change, but this increased rigidity of cartilages would not operate in any other way than by increasing and rendering more fixed the enlargement of the chest.

It is from this latter view of the mechanism of emphysema that one can appreciate with increased force the fact that it is in the lessened power of contracting the chest and emptying the lungs that the great defect lies in emphysema. The act of expiration is never completed; there is too much residual air constantly in the lungs, and inspiration is short and jerking from the act being commenced where it should end.

The force of expiration as estimated by the manometer is remarkably lessened in emphysema, whilst that of inspiration remains normal, or it may be even increased (Waldenburg);[4] the relationship between inspiration and expiration being thus, in this respect, the reverse of that obtaining in health. The vital capacity of persons with emphysema is, as might be supposed, greatly curtailed.

CLINICAL VARIETIES OF PULMONARY VESICULAR EMPHYSEMA.—Whilst pulmonary vesicular emphysema has but one essential pathology, its distribution may be local or general, and as above shown, it may vary in degree between wide limits.

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[1] Dr. Jackson of Boston, quoted by Greenhow and Jenner.

[2] Chronic Bronchitis, etc., pp. 121-122, 1869.

[3] Quain's Dictionary, 1882.

[4] Die Pneumatometrie und Spirometrie, von Dr. L. Waldenburg, 1880, p. 56.

Local pulmonary vesicular emphysema.—(1) A dilatation of the lung, accompanied by more or less atrophic changes, which takes place around old cicatrizing nodules of disease, contracting cavities, and points of pulmonary collapse from any cause. The site of the emphysema is here determined by that of the primary lesion, on which its presence is mechanically dependent. Whilst the effect of the emphysema is clinically to mask the physical signs proper to the consolidation; it brings no compensatory advantage to the patient, since the emphysematous portions of the lung are deficient in respiratory function. (2) It often happens that a whole lung is disabled by destructive disease, by permanent collapse from long-continued fluid pressure, or interstitial fibrous growth. In these cases it is inevitable that the opposite and originally unaffected lung shall enlarge, and fortunately in many instances this enlargement is really of the nature of compensatory hypertrophy, *i.e.*, there is greatly increased mobility of the side, the respiratory murmur is loud and puerile over the whole extended area of the lung, and the patient's breathing powers are fairly maintained; we may safely assume in such cases increased function corresponding with increased size, which is at least the clinical definition of true hypertrophy. It is not easy to obtain opportunities for the minute examination which is, however, I believe, only required to find a corresponding development of lung texture, with increased blood supply, and an absence of the atrophic changes of emphysema. In other cases, however, in which the lung disablement has occurred in a subject of broken constitution, whether from hereditary tendency, prolonged fever, faulty habits, or comparatively advanced age at the time of attack, the "sound" lung whilst enlarging to the clinical outline and pattern of hypertrophy, does not yield to auscultation the sounds of vigorous breathing, but a feeble *diluted* breath-sound wanting in concentrated vesicular quality. The expanded side lacks mobility; rustling crepitant sounds may be heard at different points, especially near its anterior margin. The patient has not the signs of improved aeration; he presents a livid tinge about the peripheral parts and extremities, and his enlarged lung has brought no corresponding relief to his breathless condition. Here we have a dilatation of the lung mechanically induced, and attended with atrophic changes, which constitutes emphysema.

General pulmonary vesicular emphysema.—A. *Large lunged emphysema.* This is the condition of typical, symmetrical emphysema, to the description of which this chapter has been chiefly devoted. It is only necessary further to say or rather to repeat, that there are really two groups of cases included in the variety, viz., (1) that in which the disease is distinctly secondary to recurrent bronchitis, or to chronic lung overstrain, and in which the degenerative changes supervene, and render the lesion permanent and irrecoverable. (2) That in which the atrophic changes are primary, and the bronchitis and other phenomena are secondary. Such cases are either hereditary or are induced by intemperate living, or acquired cachexia, gout, syphilis, etc.

B. *Small-lunged or senile emphysema.* This resembles the second group of the last variety in being an emphysema of essentially atrophic origin. It is, however, but the atrophy of old age most obvious at the lungs, but present everywhere. The pathology of this disease, if such it can fairly be called, is identical with that of the preceding, save that it supervenes in lungs already small and shrunken with the general atrophy of the body. Such people are prone to bronchial attacks, their heat-sustaining powers are very feeble, but under good conditions of warmth and clothing, with careful living, and a moderate amount of stimulants, they may enjoy life to even beyond the average age.

The clinical phenomena presented by cases of marked emphysema are only in comparatively small part significant of the emphysematous lung alone, being largely due to attendant and for the most part secondary lesions.

The physical signs of emphysema will be sufficiently indicated in the description of the following extreme case of the disease:

George D——, oil and tallow warehouseman, admitted into the Brompton Hospital under my care in December, 1875. The patient was a tall man with no hereditary tendency to phthisis, but of gouty parentage on the father's side. He had up to three years before admission never been laid by with any illness. At that time he had an attack of bronchitis, and since then had complained of shortness of breath and cough, constant in the winter, attended with frothy and viscid expectoration and with a sense of constriction felt below the ribs. He had never spat blood nor suffered from hectic; lost and gained flesh rapidly.

His principal symptoms on admission were, great shortness and difficulty of breathing; the head and face becoming congested, almost cyanosed at times, during attacks of dyspnœa and cough. No pain, but great sense of constriction below the ribs. Appetite bad, digestion tolerably good, bowels irregular at present but relaxed by medicine. Sleep fairly good.

Physical signs.—Pulse 86. Respiration slow and forced. Temperature normal. Great œdema of lower extremities and scrotum. Chest greatly expanded: extraordinary muscles of respiration prominently employed, respiratory movements mainly thoracic. Intercostal spaces above nipple level slightly depressed during inspiration, becoming quite level with the ribs on expiration. Below nipple level intercostals greatly depressed during inspiration, becoming level with ribs or even slightly puffed outwards during expiration; seventh and eighth ribs yield inwards with inspiration. Heart's impulse most perceptible at left costal margin at the level of the tip of ensiform cartilage.

Girth of chest on each side above nipple level, 18 inches. At level of base of ensiform cartilage right side, $18\frac{5}{8}$, left $18\frac{1}{2}$ inches, $\frac{1}{4}$ inch extreme expansion with inspiration in both these situations. The whole chest resonant down to the margin of the ribs both in front and behind; at posterior bases on both sides fine, bubbling râles heard principally with inspiration. Similar râles heard over lower two-thirds of the right and left fronts. Apices clear, cardiac sounds unattended with bruit. Abdomen somewhat distended, and contains a small quantity of fluid. Liver depressed.

The subjoined tracings show very well the nature of the respiratory movements in this case. They were taken by a very simple apparatus consisting of a straight rod connected by a flexible joint with an expanded button to apply to the chest, the other end scratching upon a horizontal sphygmograph plate previously smoked.

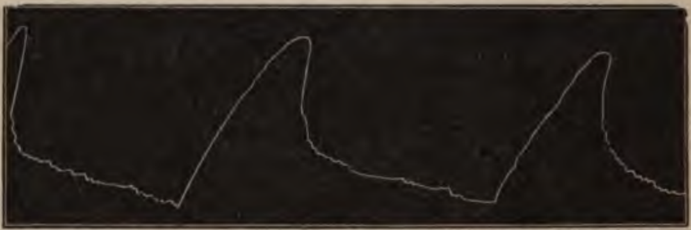

FIG. 13.—Tracing of respiratory movement showing total forward thrust at third mid-sternum.

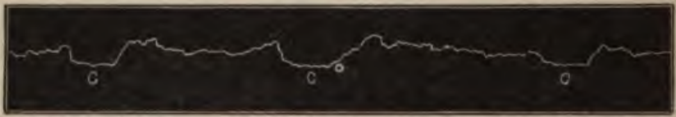

FIG. 14.—Tracing, showing recession, C, C, C, during inspiration, seventh rib, axillary line.

Tracing Fig. 13 represents the movement of the sternum at the level of the third cartilage. (The man was sitting in a chair, with his back resting against a flat board). It is equivalent to exaggerated thoracic breathing, although the patient was inspiring in the degree natural and necessary for him. Tracing Fig. 14 was taken at the 7th rib in the lateral region, right side, and shows a distinct recession *c*, *c*, *c*, during each inspiration.[1] This was perhaps due to the bases of the lungs being in some measure disabled by secretion, but the disablement and collection of secretion were undoubtedly in the greatest measure owing to the inaction of the diaphragm in consequence of the flattening of its arch. The thoracic movement was certainly somewhat in excess in this case from the same cause, but in most cases of emphysema the respiration becomes thoracic rather than abdominal.

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[1] The right lung was found post mortem to be more highly emphysematous than the left.

The patient died, after he had been in the hospital three weeks, from general dropsy and cyanosis.

The *post-mortem* examination revealed the usual phenomena of large, dilated heart, the right side most affected, the tricuspid orifice measuring 6½ inches in circumference; large emphysematous lungs, the emphysema most marked at the anterior and upper parts and in the right lung, there being œdema and slight congestion at both bases; there were no signs of active bronchitis; the bronchial tubes contained a frothy thin fluid; spleen hardened, kidneys mechanically congested, liver enlarged and fatty.

TREATMENT.—It will be obvious from what has preceded that the treatment of emphysema is mainly palliative, consisting of the prevention of fresh catarrhs, asthma and bronchitis, the avoidance of over-exertion and straining occupations, the escape from dusty, irritating atmospheres, and, when possible, the timely migration to more genial climates during the winter or early spring months.

We cannot cure emphysema, but we may by judicious measures arrest textural decay, and prevent fresh over-strain. Emphysema, let me again remark, is never, within the normal period of human life, fatal, save in its complications, but it is the factor which endangers recovery from many diseases. In the *dietetic* treatment of emphysema, we must so far restrict ingoings as to adapt the resulting products:—

1. To the needs of a necessarily limited muscular activity, a lessened tissue metamorphosis.

2. To a somewhat retarded circulation through the lungs from capillary obliteration.

3. To a lessened oxygenation and a corresponding tendency to plethora of venous blood.

A restricted, well-assorted dietary, and the maintenance in fair activity of the eliminatory functions of the skin, kidneys and bowels, will fulfill the double indication of avoiding surcharge of the economy by waste materials, and diminishing the tendency to venous plethora, visceral congestion, and over-work of heart.

With regard to the treatment of emphysema by drugs, there are certain very clear indications to be followed. Measures for the regulation of secretions have been alluded to, and require no further detailed description; they will from time to time be called for by the disordered condition of these secretions.

Intercurrent attacks of bronchitis, asthma, etc., will require their appropriate treatment, but except at such times one should—so to speak—forget the lungs in the medicinal treatment of this complaint. Dyspnœa is not always to be regarded as an indication for ether nor a bronchial wheeze for squills. We must rather have careful regard to the general condition of the patient, and especially to vessels and heart tonicity.

Iron, arsenic, and strychnia are the best general tonics, and should be given in small doses for lengthened periods, say for a month at a time with intervals of rest from drugs. The arseniate of iron is an excellent preparation for our purpose, *e.g.*, in $\frac{1}{12}$ to $\frac{1}{8}$ gr. doses with $\frac{1}{2}$ gr. of nux vomica, and a little pepsin twice a day after food. A little aloes may be added if necessary, or the occasional morning use of aperient waters with more rarely a mercurial, with the view of maintaining equilibrium in the portal system. Five drop doses of tincture of iron with a little strychnia, twice a day, will often prove of value in restoring muscular tone to the heart and to the bronchi, after any fresh attack of bronchitis or asthma, involving renewed strain upon the right ventricle; and occasionally at these times it is desirable to give digitalis in moderate doses for a few weeks, five minims three times in the day is usually quite sufficient for the purpose.

In the advanced stages of emphysema and its concomitant affections, when the limbs become dropsical, the abdomen full, the viscera engorged, and with—what is the key to the whole situation—the right ventricle fluttering at the epigastrium, the pulse small, irregular and intermitting, and the jugulars distended and filling from below; in these cases absolute rest in bed, the free administration of digitalis, with diuretics and diffusible stimulants, will sometimes still serve to rescue such patients from impending death. The flow of urine freely returns, the pulse steadies, and the dropsy subsides under this treatment. Albuminuria, usually more or less present under these conditions, is no contra-indication to the use of blue pill, which, given in combination with squill and digitalis for three or four successive nights, will frequently give a start in the direction of improvement.

The heart is the failing link in the phenomena present, and digitalis is the remedy, but it sometimes taxes our ingenuity and resolution to give the drug in a combination in which it can be borne. Convallarin may be tried as an alternative sometimes, but is by no means equal to digitalis, in influencing heart and vessels.

The true value of the employment of compressed or rarefied air in the treatment of emphysema, has not yet been fully ascertained. But in endeavoring, so far as is possible, to form a judgment upon the subject, we must carefully distinguish between condensed or rarefied air climates or baths, and the same modifications of air pressure brought to bear upon the interior of the lungs only, not upon the whole body. If a man, himself surrounded by the ordinary atmosphere, inhales from a compressed air chamber, his lungs become forcibly expanded by an air rich in oxygen on account of its concentration. Such a treatment may be useful in certain cases of collapse from long-continued compression of the lung by fluid or from inflammatory thickening, but it is not adapted for a patient whose chest is already over-expanded from impaired power of expiration.

The effect, too, of inhaling compressed air in this way is to render the lungs more anæmic, whereas, in emphysema, they are already defectively supplied with blood; and the only advantage, on the other hand, to be gained is the doubtful one of displacing the stagnant residual air by one highly charged with oxygen.

If, however, the emphysematous patient be immersed in an atmosphere compressed, say, to half again the density of ordinary air, his respiratory mechanism is neither helped nor impeded by the new conditions, for the atmospheric pressure is not only increased on the inner surface of the lungs, but also, and similarly, over the whole body. He is simply breathing a concentrated air, but as the vital capacity of his lungs is considerably diminished, some advantage may be thus obtained from the air inhaled being (bulk for bulk) richer in oxygen. Be it remembered, however, that with increased density we get diminished *mobility* of the air particles, so that the osmotic interchange of gases on which respiration truly depends, is more slowly effected. Again, the heart's action is somewhat impeded by the increased pressure upon the vessels; still it is true that emphysematous people do well at the sea-level, and I have found at the Brompton Hospital, where a compressed air-bath has recently been fitted, that patients with emphysema experience in the bath decided comfort, and are perhaps somewhat benefited by its use. These baths cannot in any sense be regarded as curative in emphysema.[1]

It is generally held that elevated climates are unsuitable for patients with emphysema, inasmuch as such patients must breathe more deeply to obtain the same amount of oxygen from a rarefied atmosphere. Respiration in a rarefied air would thus tend rather to increase the evils already present in an over-expanded chest with deficient power of expiration. But there are some considerations from which it would appear that such an atmosphere is much less unsuitable to emphysematous patients than one might at first assume.[2]

(*a*) In the first place it must be remembered that, with an abundant air supply, we use only a very small proportion of the oxygen present in the air for our respiratory purposes—in other words, expired air is not nearly exhausted of its oxygen; (*b*) secondly, although the air of elevated regions is rarefied, its particles are more actively mobile, and oxygenation is relatively quickened. Thus Tyndall and Frankland[1] have shown that the loss of weight of a candle burning on Mont Blanc at an elevation of 1200 feet, is identically the same as that of another candle of similar dimensions, burning in the valley of Chamouni below; (*c*) thirdly, the circulation through the lungs, as elsewhere, is carried on at less pressure in elevated regions, and the heart, tuned originally to lower latitudes, finds relief in this way.

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[1] Vide Walshe, Diseases of the Lungs, 4th edit., p. 333. Braun, The Curative Effects of Baths and Waters, edited by H. Weber, M.D., p. 53. Cohen, Inhalation in the Treatment of Disease, Philadelphia, 2nd edit., p. 41. The simplest compressed air-bath consists of an iron chamber, into which air is gradually pumped to an excess of half an atmosphere or more. The bath is in daily use at the Ben Rhydding Sanatorium.

[2] It has been proposed to use rarefied air-baths, but I need scarcely say that the above remarks refer exclusively to the rarefied air of elevated regions, inasmuch as one would hardly with seriousness suggest placing any patients under an air pump!

There are many other features, besides the mere elevation, with which we are now only concerned, which would of course have to be taken into consideration in coming to a decision as to the relative advantages of high or low climates in the treatment of emphysema. No statistics upon the subject have, so far as I know, been published.

But, inasmuch as it is the *expiration* which is at fault in emphysema, and since, from the defective lung recoil, there is an undue amount of residual air retained in the lungs, the most decided treatment, indeed the only real treatment of the physical kind under consideration, is that of causing the patient to expire into a partially exhausted chamber. This method of treatment has been tried by several good observers, especially by Waldenburg, Schnitzler[2] and Berkart.[3] Dr. Waldenburg[4] relates several cases in which marked benefit has ensued upon the employment of this treatment; and although most of his results are complicated by the simultaneous use of turpentine, saline spray or other inhalations, yet the relief of symptoms and gain in vital capacity cannot by these other means be accounted for. In most of his cases, Dr. Waldenburg caused the patient to inspire compressed air and to expire into rarefied air.[5]

These mechanical methods of treatment are attended with many practical difficulties, and the results obtained are only as yet sufficiently encouraging to warrant fresh investigation. And further, it must always be carefully remembered, that we have in emphysema to deal with *a more or less advanced degenerative disease of the lung texture.*

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[1] Quoted by Braun, loc. cit., p. 59.

[2] Wiener Klinik, 1875, Heft 6. See abstract of Dr. Schnitzler's views in Dr. Dobell's Reports, vol. ii., p. 114.

[3] Lancet, Nov. 25th, 1871.

[4] Die Pneumatische Behandlung der Respirations- und Circulations-Krankheiten. Berlin, 1875, pp. 385-411.

[5] Waldenburg's apparatus is an adaptation of Hutchinson's spirometer, with weights so adjusted as to exercise positive or negative pressure upon the contained air as may be required. It is described and figured in his work (p. 128), and also in Cohen's work (p. 45).

CHAPTER IX.

ŒDEMA OF THE LUNGS.

Œdema of the lungs consists of an escape of serum from the vessels into the interstices of the organ, and into the alveolar and bronchial spaces.

There are many causes of this condition, but they range themselves naturally under two heads. 1. Disturbance of circulation. 2. Morbid conditions of blood.

Thus, we may have *inflammatory œdema* from active congestion, and *mechanical œdema* from retarded circulation, which may be due to mere feebleness of heart, or obstruction to the passage of blood through the lungs, as in emphysema, mitral stenosis, mitral regurgitation, or pressure from tumors on the pulmonary veins. A certain amount of œdema, due to impaired vascular tonicity, commonly remains for some time after acute inflammation of the lung has passed. The powerful inspiratory efforts to draw air into the lungs through the constricted passages in croup and asthma, and other extended bronchial obstructions, cause an afflux of blood resulting, in some cases, in more or less œdema of the lung textures.

In all those conditions of blood which favor the escape of serum from the vessels we may have pulmonary œdema. Of these conditions albuminuria is the most important; scurvy, purpura, anæmia, hydræmia, come a long way afterwards as possible causes.

Morbid Anatomy.—Œdematous lungs are large, heavy, wet, indented by the ribs, pitting on pressure, and on section exuding from their texture and tubes an abundant, frothy serosity, sometimes blood-stained. All these characters are especially marked at the bases, or most dependent parts of the organs, and indeed, may only be apparent there unless otherwise locally determined. Œdema affects both lungs, but most commonly one side is more affected than the other, owing to the posture adopted by the patient. It is very usual to find the pleura on one or both sides containing an undue amount of serum. Except at the extreme bases, and when very thoroughly water-logged by old standing œdema, the lungs are more or less crepitant, and portions cut off will float in water. Under the microscope some epithelial shedding may be observed in the alveoli; otherwise the texture of the organs is unchanged by œdema alone.

Symptomatology.—The *symptoms* of œdema of the lungs are mingled with those of the other diseases of which this condition is but a consequence. Dyspnœa, straining cough, with thin, watery, mucoid sputa, are the chief symptoms. The patient sits up supported in bed, the respiratory movements are thoracic, "lifting" in character, the bases of the chest receding with inspiration. The front of the chest is hyper-resonant, the posterior bases more or less dull. Over the posterior aspect of the lungs, extending from the base upwards, the respiratory sounds are enfeebled or annulled, and fine, bubbling râles are heard chiefly with inspiration.

Emphysema crackle, pneumonic crepitation, and the crepitation of air penetrating a collapsed lung, are of almost identically similar characters so far as the *râle* is concerned, with that of œdema. The distinctions must be drawn from associated percussion and auscultatory phenomena.

PROGNOSIS.—Œdema of the lungs is usually of grave significance. In chronic Bright's disease, and in cardiac dropsy it is one of the later phenomena. In those forms of heart disease, however, telling directly upon the circulation through the lung, a certain degree of pulmonary œdema may precede general dropsy, and may long persist without further consequences. It is very common in old people with feeble hearts and emphysematous lungs, to have a certain amount of pulmonary œdema as a permanent condition, and I have met with several people in whom slight œdema sounds have for years persisted on one side, probably the result of a past inflammatory attack. No doubt some of these cases are better explained by an œdematous condition of the connective tissue uniting the pleural surfaces over a lung that has been the seat of a former inflammatory attack. The supervention of œdema of the lungs in acute bronchitis, or in pneumonia, is of very fatal augury, being due to failure of heart.

TREATMENT.—The treatment of acute œdema from failing heart is referred to in the chapter on pneumonia. In cases of local œdema from loss of tone of vessels after inflammation, iron, mineral acids, and sometimes small doses of digitalis are of great value.

In all other serious cases of pulmonary œdema, derivative treatment is called for. Dry cupping gives much relief, especially in obstructive cardiac disease, and in emphysema with dilated heart.

The vegetable diuretics, juniper, scoparium with digitalis, and moderate doses of the iodide and neutral salts of potash, are valuable; watery purgatives and diaphoretics are to be used in turn. The exact nature of the case, whether renal or cardiac, will determine us in selecting our remedies. In cardiac cases we rely more upon diuretics and digitalis, with occasional small doses of mercurial and saline aperients. In renal cases our derivative treatment is rather effected through the bowels and skin, by brisk, watery aperients and sudorifics, including air-baths. In all cases we must fairly support the patient.

CHAPTER X.

PNEUMONIA.

PNEUMONIA may be defined as an acute febrile disease, characterized by inflammatory consolidation of some portion of one or both lungs.

It is true in a sense that pneumonia and inflammation of the lungs are synonymous terms, but the latter expression by no means covers the whole pathology of the disease, indeed a careful examination of the etiology and clinical features of pneumonia, would suggest its being placed in our nosology amongst such diseases as acute rheumatism, erysipelas, quinsy, influenza, the inflammatory phenomena in each of these diseases being but the local expressions of a general state, and only exceptionally proceeding beyond hyperæmia and simple exudation.

ETIOLOGY.—(*a*) *Individual predisposition.* No age is exempt from pneumonia, but the disease occurs most at the period of life between twenty and forty, when persons are most exposed in the active struggle of life. The male sex is more frequently attacked than the female in proportion of three to two, and at the period of life referred to, the prevalence is twice as great amongst males.[1] Depressed vitality, arising from debauchery, intemperance, over-fatigue, anxiety, insufficient food, over-crowding, renders the individual more prone to attack.

(*b*) *Previous diseases.* Chronic disease of any kind, but especially alcoholism, albuminuria, and gout, are liable to engender pneumonia. A plethoric state of body seems also to favor its occurrence, and to add much to the severity of attack.

(*c*) *Climatic influences.* Cold seasons, great variations of temperature, rough, cold winds, bring about pneumonia, and account in many instances for its epidemic prevalence. Dr. Hjaltelin considered a highly ozonized condition of atmosphere, which he observed to exist coincidently with the Iceland epidemic described by him, to have had something to do with its occurrence.[2]

(*d*) *Epidemic influences.* An epidemic of influenza has in some instances preceded a similar occurrence of pneumonia.[3]

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[1] Memorandum on the incidence of Fatal Pneumonia, by G. B. Longstaff, M. B., Oxon., F.S.S., drawn from the Registrar General's returns for the decade 1871-80. Collect. Invest. Record, vol. ii., July, 1884.

[2] Epidemic pneumonia in Iceland in the year 1863, by John Hjaltelin, M.D., Inspecting Medical Officer of Iceland. Edin. Med. Jour., vol. ix., 1864, page 969.

[3] Hjaltelin, loc. cit.

Ziemssen,[1] on a careful consideration of the occurrences of pneumonia, which have been tabulated for Europe, Northern America and Africa between the years 1836 and 1856, finds that the curve of pneumonia fluctuations is not concentric with that of other inflammatory diseases, whilst it remarkably coincides with the curve of typhus.

Exciting causes of Pneumonia.—Cold, either in the form of absolute depression of external temperature, or chills from undue exposure to cold draughts, insufficient clothing, damp beds and the like, is by far the most common exciting cause of pneumonia. Not only are most cases of sporadic pneumonia to be thus accounted for, but a considerable proportion of the epidemic occurrences of the disease in modern times are attributed to lowness of temperature and cold winds; individual susceptibility and special exposure being the predisposing circumstances. Thus, the epidemic in Iceland in the winter of 1863, was prepared for, by a previous occurrence of influenza, presumably rendering the respiratory organs vulnerable, and was attended with rough and cold weather, and winds highly ozonized. The epidemic related by Dr. Welch,[2] as affecting the 22nd Regiment stationed at New Brunswick, was similarly explained. From a strength of 652 men, 52 were attacked; 12 cases occurred in January, the coldest month; 32 in February and March, the months of greatest temperature fluctuations; 320 of the men were housed in the "exhibition building," a large, cold, draughty, wooden structure, freely exposed on all sides; of these 38 were attacked. Of the whole 52 cases, Dr. Welch attributes 27 to lowness of temperature and cold draughts, 7 cases to sleeping on mattresses carelessly stuffed with damp, snowed straw, 5 to exposure to great cold at night, 6 to chilling of body whilst perspiring from strong exercise, and in 7 cases the exciting cause was obscure. Dr. Welch does not consider that climatic conditions were operative, save in the one factor of low temperature. Intemperance seems to have been present as a predisposing cause in many instances, and it is further stated that the men had had a previous six years' service in Malta. Out of 152 women and children, who had in this latter respect been under the same conditions, however, only two cases of pneumonia occurred—a remarkable circumstance, attributable no doubt to less exposure, and superior shelter in the warmer barracks and married quarters. It is to be observed that merely a low external temperature may not be so favorable to the occurrence of pneumonia as exposure to great variations. It frequently happens that workmen getting warm at their work in the sunshine of a May-day throw off coverings, and get chilled by a N. or N.E. wind, of which they were before unconscious.

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[1] On the fluctuations in frequency to which pneumonia is liable, with special reference to those occurring during the two decimal periods, 1836-1856, Prager Vierteljahrschrift, 1858, Bd. ii., and Edin. Med. Jour., vol. iv., 1858.

[2] Army Medical Reports, 1867, Appendix ix. Remarks on Pneumonia.

Septic causes.—Comparatively recent etiological researches, and especially the evidence brought together by the Committee of Collective Investigations, leave no room for doubt that cases and groups of cases of pneumonia are occasionally met with, which are attributable to bad sanitary conditions, and especially exposure to sewer gas emanations.[1] The disease when thus arising is of somewhat different type from the inflammatory form, and merits the name "pythogenic"[2] that has been used to distinguish it.

Other cases occur which may be classified under this heading, and which are more distinctly secondary to septic poisoning, such as the pneumonias which complicate general septicæmia, induced by fœtid absorption from unhealthy wounds.

Infectious pneumonia.—There is also evidence to show that, under certain circumstances not completely known, pneumonia may be communicated by one person to another. The Collective Investigation Committee, in answer to a special request for information as to the etiology of pneumonia, received about 100 replies, in eighty of which no other cause than exposure to cold could be assigned for the attack. The twenty remaining replies are abstracted in the Record,[3] and amongst them nine observations are included, in which there was apparent transmission of the disease from one member of a family to others, in at least one of which the evidence is very strong. The difficulty is of course the usual one, of separating cases of several persons being attacked in consequence of exposure to a common cause, from those in which the first attacked has transmitted the disease to others. It is probably the pythogenic form of the disease, in which infection is most to be met with, but not exclusively so. In the epidemics at Iceland and New Brunswick, already referred to, no suspicion of communication of the disease by infection is mentioned, and similarly with one of the two epidemics described by Surgeon-Major Maunsell[4] in N.W. India (Mooltan and Bunnoo), in 1882 and 1883, both of which were apparently due to conditions of temperature. In the second epidemic, however, twenty of the attendants were attacked.

Pneumonococci.—Any statement with regard to the etiology of pneumonia would be far from complete without careful reference to certain micro-organisms which are held by some observers to be the originators and propagators of the disease.

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[1] The admirable reports on Epidemics of Pneumonia, British and Foreign, by Dr. Sturges and Dr. Coupland in the second (1884) volume of the Collective Investigation Record, part i., are the most recent, and contain full literary references on this subject.

[2] A term first suggested by the late Dr. Murchison for typhoid fever, Med. Chir. Trans., vol. xli., p. 221, and first applied to pneumonia by Drs Grimshaw and Moore, Dublin Journal of Medical Science, 3rd series, vol. lix., 1875.

[3] Vol. ii., page 60, et seq.

[4] Collective Investigation Record, 1884, pp. 77 and 93.

Friedländer (Virchow's *Archiv,* Bd. 87) was the first to recognize the constant occurrence of masses of micrococci in lungs affected with acute croupous pneumonia. They are oval in shape, about 0.001 mm. long, and may occur singly or as diplococci (dumb-bells) or in chains. One observer, Ziehl, attributes the characteristic brown sputum to the presence of numbers of these micro-organisms.

Dr. Giles, writing in the *British Medical Journal,* July 7th, 1883, found the micrococci both in the blood and sputum in cases of pneumonia occurring in India. This observer produced pneumonia by injecting the cultivated organism into the subcutaneous tissue of rabbits.[1]

Dr. Klein[2] mentions that Friedländer and Frobenius, also Salvioli and Zäslein have produced pneumonia in mice, rats, and rabbits by inoculation from cultures.

Klein has investigated the matter, and finds besides bacilli other species of micrococci present. In the lungs of patients dying of pneumonia he found the organisms, but in many of the alveoli he found fibrin and blood corpuscles but no micrococci. He regards the dependence of the disease on these micrococci as questionable, for he found injection of the cultivated micrococci into rabbits and mice produced septicæmia but not pneumonia. The peculiar hyaline capsule, which Friedländer maintains is characteristic of this form of pneumonococcus, Klein has seen round the micrococci in pyæmia of rabbits produced by inoculation from swine plague (schweinesuche).

Some years must yet elapse before the exact position of micro-organisms with regard to disease can be finally determined; and, before this particular micro-organism can be accepted as the spore of pneumonia, *i.e.,* as the only means by which that disease can be originated or propagated, there are many difficulties to be surmounted. The climatic conditions in which the disease most occurs, its common origin in exposure to surface chill, the rareness with which infective propagation is observed, and the not yet ascertained constancy of the presence of the coccus, nor of its virulence under cultivation, are circumstances requiring to be explained by the exclusive germ theorists of pneumonia. It is a point worthy of note that the diseases already enumerated as those with which pneumonia may be best compared, are equally remarkable for their apparently double mode of origin, viz., from exposure to chill, and from exposure to poison influences. One might be tempted to say that each of the two diseases, ordinary and pythogenic pneumonia, or idiopathic and wound erysipelas, or influenza and virulent catarrh, is quite as distinct from the other as typhus is from enteric fever, but clinical experience does not favor this view. To take a simple instance, a slight dampness of feet will with almost unerring certainty give to many persons a severe catarrh with the usual symptoms of malaise, chilliness, coryza, etc. The catarrh thus acquired is virulent, and will run through the household. The composite organic "splinters" from the first subject who acquired the catarrh, *may* demonstrably contain micro-organisms to account for their contagiousness, yet the onset of the disease can scarcely be attributable to the reception of such organisms, but must rather be regarded as some form of vaso-motor disturbance bringing about changes, possibly favorable to the habitation of ever-present germs. In considering these difficult but interesting and important problems, one must not confound what may be the carriers of the disease with the disease itself, nor look upon what *may* be only *epiphytes* as genuine *disease-spores*.

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[1] Dr. Dreschfeld in a paper communicated to the Medical Society at Manchester, 1884, describes a peculiar form of pneumonia which had been recently prevalent there, intermediate in character between ordinary pneumonia and the septic or "epidemic" form of the disease. Micrococci were abundantly found post mortem in the exudations, pericardial effusions and blood. Dr. Dreschfeld regards the different forms of pneumonia as varieties of one disease, due most probably in all cases to a specific infective agent, the pneumonococcus. British Medical Journal, May 10th, 1884. See also Ueber Wanderpneumonie und ihre Beziehung zur epidemischen Pneumonie, von Prof. J. Dreschfeld. Fortschritte der Medicin, Bd. iii., 1885.

[2] Ein Beitrag zer Kenntniss des Pneumonokokkus, in Centralblatt für die Med. Wiss., Berlin, no. 30, July, 1884.

PATHOLOGY AND MORBID ANATOMY.—The morbid anatomy of pneumonia consists of an acute hyperæmia of the affected lung, resulting in a fibrino-corpuscular exudation into the alveoli, perhaps including the smaller bronchioles, and forming a film upon the pleural surface. This exudation coagulating *in situ*, fixes the lung in a state of immoveable expansion more densely solidified than it could be by any artificial injection with coagulable fluid. In the accomplishment of this second stage of "hepatization," all the severity of the disease is manifested, and with its attainment the disease proper is at an end.

The local consequences of the disease do not thus terminate, however. With the first consequence of extraneous influences—coagulation of the exuded products—a chain of secondary phenomena commences, partly chemical, partly vital, attending their liquefaction and re-absorption. Thus, after a usually very short, but sometimes more prolonged period of malaise, the malady is ushered in abruptly with rigors, rapidly mounting and maintained high temperature, on the second to the fifth day of which the local signs present themselves; on the seventh to the tenth day the temperature falls, but the local lesion has yet to be removed.

Pneumonia is thus artificially divisible into three stages.

The first stage begins with the rigor and ends with the appearance of definite signs of consolidation. It may be termed the stage of initial fever with pulmonary engorgement, and it lasts from two to five days. If death should occur at this stage, the affected lung is found to be in a condition of inflammatory œdema, heavy, engorged with florid blood, pitting on pressure, and still crepitating; on section, exuding abundant, blood-stained, frothy serosity. Pulmonary hyperæmia and fluid exudation are the conditions present. When the pneumonia occurs in very cachectic subjects, or in its more intensely pythogenic forms, scattered pulmonary hemorrhages may be found. A mere out-burst from engorged vessels at some one time, however, is not necessarily associated with disease of a specially low type. It must be observed that throughout the febrile period of pneumonia there is an advancing area of lung thus affected.

The second stage, that of pulmonary hepatization, emerges from the former and terminates in from forty-eight hours to five or six days. It is characterized by continued high temperature and by increasing signs of consolidation of lung.

In this stage of *red hepatization* the affected portion of lung is bulky, heavy, and solid to the feel. The pleural surface is covered with a thin layer of soft, finely granular, lymph, which can be readily scraped off, exposing the glistening pleura beneath. On section the lung is firm and dry, presenting a red granular surface which is readily broken by the pressure of the finger. There may be a little frothy secretion in the bronchial tubes, the mucous membrane of which is injected. A portion cut from the consolidated lung sinks at once in water. A thin section of a portion of the consolidation when microscopically examined shows the alveoli, alveolar passages, and sometimes the smallest divisions of the bronchi, to be occupied by closely packed red blood discs and leucocytes entangled in the meshes of coagulated fibrin. The alveolar wall is not changed beyond sometimes slight swelling of its epithelium, a few cells of which may be shed. Towards the latter portion of this stage there is more or less emigration of leucocytes from the alveolar walls. In cases in which the pulmonary inflammation supervenes upon hypostatic congestion in continued fever, the consolidation is softer, darker, and more spleen-like in appearance and has been called "*splenification of lung.*"

Third stage.—That of resolution. The commencement of this stage is characterized in normal cases by a remarkably rapid fall of temperature attended with profuse sweatings or other critical phenomena, and the signs of commencing liquefaction of the elements of the lung consolidation. This stage of pneumonia is also termed that of *grey hepatization* in its earlier period, and sometimes by the more erroneous term *purulent infiltration* at its later period. The exuded products now rapidly undergo molecular degeneration, the fibrinous element becoming completely emulsified, and the corpuscles more or less broken down by fatty changes. As a rule the alveolar walls remain intact, beyond perhaps slight swelling of their epithelial lining, and the emulsified products are more or less readily removed, chiefly by absorption, but, in part, also, by expectoration.

In some cases, however, the texture of the lung is involved in the inflammatory change, the alveolar walls become infiltrated with leucocytes, and the reparative stage of grey hepatization is changed for that of advancement to suppurative disintegration of the lung itself. This untoward event must be regarded as a complication rather than as a feature of pneumonia.

It is again not difficult to understand how, in course of the dangerous processes above described, thrombosis of larger branches of the pulmonary artery may take place, or such extensive capillary stagnation as shall lead to necrosis and abscess or gangrene of a portion of the lung.

The portion of lung affected in pneumonia is most commonly the base on one side, the right being more frequently affected than the left. Both bases are rarely simultaneously attacked, but it is not very uncommon for the second base to become more or less involved in the course of the disease. Perhaps the frequency with which this happens has been exaggerated from the fact that both tubular breath-sound and crepitant râle may be audible at one base by reflection from a corresponding point of the other.[1] The upper lobe is attacked in a goodly proportion of cases, more especially so in children than adults; although, amongst the latter especially, apex pneumonia includes the more cachectic cases, from alcohol and other causes, yet the disease in this situation otherwise runs a normal course.

SYMPTOMATOLOGY.—The general aspect and symptoms of pneumonia on the third day of attack are usually such as to leave but little room for doubt as to the nature of the case. The flushed look and burning skin, the hurried noiseless breathing, and rapid but regular pulse, the frequent short cough half-stifled from pain, the dryish thickly-coated tongue, and the singular prostration of the patient, are a group of signs which, supervening speedily upon a well-marked attack of shivering, cannot be otherwise interpreted than as being those characteristic of an attack of ordinary *acute basic pneumonia* in full intensity.

On closer examination the respirations are found to number about 40 in the minute: they are not obstructed nor attended with such marked action of the nares as in capillary bronchitis. The pulse 120; the temperature 104°. Fig. 15. On inspecting the chest its movements are found to be chiefly one-sided, but there is no apparent difference in size on the two sides, and the heart is found beating in its normal position. The movements of the affected side are voluntarily restrained by the patient, and a severe pain referred to this part often cuts short the cough or any attempt to draw a full breath. The percussion signs in front are not materially altered; on auscultation the respiratory murmur is found here to be weakened on the affected, exaggerated on the healthy side. Posteriorly, over the base of the affected side the percussion note is dull, but not without some wooden quality of tone quite distinct from the dead, flat note of effusion. The dullness extends upwards to a variable height, and over the dull portion the respiration is characteristically bronchial or tubular, the voice-sounds well-conducted, broncho-phonic, and the peculiarly explosive, fine, inspiratory crepitation of pneumonia is heard, especially towards the upper limits of dullness. If there be any expectoration it is scanty, viscid, frothy, and more or less rust-colored from blood staining; it contains an excess of chlorides, and is deficient in phosphate of soda. The urine is scanty and concentrated, deficient in chlorides and sometimes yields a thin cloud of albumen.

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[1] Vide previous edition On Consumption, etc., 1878, p. 208.

I have for convenience of description taken the symptoms and signs of a typical pneumonia at its most striking period, (the second or third day after the rigor), that at which the practitioner is most commonly called upon to see such a case. There is no doubt that the general symptoms may precede the appearance of physical signs by a very perceptible interval of time, during which, on examining the chest, no dullness is as yet to be found. The breath-sound at one base has, however, at a very early period a peculiar, rough, harsh quality, very like that of exaggerated breathing, and slight crepitus may be audible. A few hours later the characteristic pneumonic crepitus is abundantly evident, whilst the percussion note, although shortened and heightened in pitch, is, as yet,

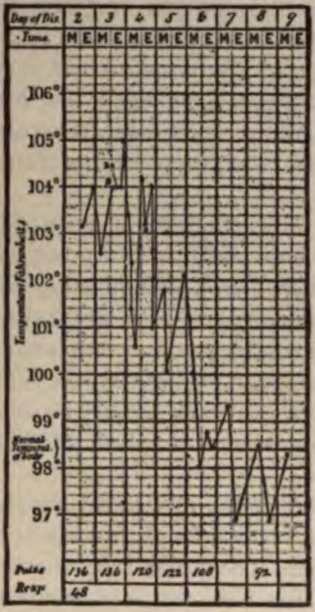

FIG. 15.

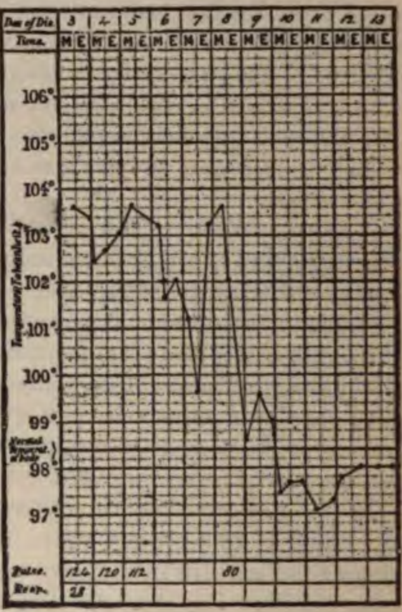

FIG. 16.

FIG. 15—The chart illustrates temperature record of a case of pneumonia (left lung), arising from exposure to chill in a child aged seven years. The physicial signs developed at the left posterior base and anterior apex on the third day. The early temperature was somewhat moderated by tepid spongings (s), and a dose or two quinine (Qx). (Middlesex Hospital Records, No. 02, 1882).

FIG. 16.—Temperature chart from case of idiopathic erysipelas, a man aged 32 admitted on third day of attack. The chart is introduced merely for comparison with that of pneumonia.

by no means dull. In other cases, again, the physical signs of complete consolidation may develop so rapidly as to render no previous stage noticeable. And here it is well to remark that there may be observed in different cases some diversity in the general symptoms which precede the pulmonary lesion. The rigors may be strongly marked and speedily followed by severe headache, and even somewhat violent delirium, such as to suggest meningeal inflammation. But the hurried *regular* breathing must not escape notice. In other cases, a marked icteric tinge of skin with gastric disturbance suggests the oncoming of jaundice, but the fever runs too high for simple jaundice, and the breathing and pulse also betoken a more acute disease. Finally, the local symptoms, severe pain and dyspnœa, may be those which chiefly attract attention, and these are not infrequently cases of the worst augury as regards the lungs—cases in which the disease is in the smallest sense idiopathic, and has rather been, so to speak, forced upon the patient by exposure to cold.

To follow our assumed case of pneumonia further onwards:—the temperature rising after the onset with rigor to 104° hovers thereabouts with but little variation (unless influenced by treatment) for four, five, six, or seven days, and then rapidly falls within forty-eight hours to normal. This abrupt fall of temperature, which marks the *crisis* in pneumonia, is often attended with very profuse sweats, a copious discharge of urine, depositing abundant lithates, and more rarely with diarrhœa. More or less exhaustion is always present, and sometimes the collapse is very marked. After this brief period of shock has passed, the patient who has been suffering throughout from complete anorexia, thirst, restlessness, and increasing weakness, with dyspnœa, troublesome cough, and blood-stained expectoration, is now rapidly relieved from many of these symptoms. The sense of dypsnœa is greatly lessened, although the breathing is still double-quick. The pulse becomes quiet, the skin moist, and the tongue begins to clean at the tip and edges. Appetite does not yet return, but the sleep is quiet and refreshing.

On examination, however, the physical signs will be found to have but little changed, dullness and bronchial breathing being as distinct as ever. At the upper boundaries of the consolidation, the crepitation if present, or when it returns (for it often is but little marked or disappears for a few days), is found to have altered in character. It is larger, moister, less explosive, and is heard during expiration as well as inspiration, although still most abundantly with inspiration. This *sub-crepitant râle* marks the commencing resolution of the hepatized lung, and extends downwards as the consolidation slowly melts away. The sputa now become more or less opaque, although usually still scanty. In some cases the expectoration at this stage is muco-purulent, amounting to several ounces during the day. But it yields no lung-tissue to microscopic examination after boiling in caustic soda solution. Sometimes, again, from first to last there is no expectoration in pneumonia.

After the period of crisis there is in some cases a slight return of fever, but of a different character to that of the original disease. The temperature assumes a hectic type, with a moderate daily rise to 101° or even 102°, and in these cases slight daily chills are complained of. These symptoms are somewhat alarming, and suggest the possibility of some caseation or softening of lung-tissue proceeding, or of purulent effusion, into the pleura. But they do not either of them necessarily bear such untoward meanings. I have observed such symptoms now in several cases in which the pulmonary lesions entirely cleared up.

Their rationale is to be sought undoubtedly in the too rapid absorption of inflammatory products resulting in an overdose of material not altogether aseptic and inert. In some cases it is possible that more pronounced local or general results may ensue. Parkes observed, "the exudation . . . may contaminate the blood during softening to such an extent as to lead to renewal or increase of the fever and inflammation of other parts; or to coagulation of the blood in the heart or great vessels."[1] Other authors, so far as I am aware, are silent on this point.

Pythogenic pneumonia.—The following case is introduced as an example of a fairly typical case of this variety of pneumonia.

Minnie L——, aged nineteen, a nursemaid living in London, a light-haired, well-nourished girl, was admitted into my ward, Nov. 27th, 1882. She had a family history of chest delicacy, but had herself had no previous illnesses. Several persons in the house where she resided, however, were stated to have suffered from feverish and abdominal symptoms. The water, of which patient drank freely, was regarded with suspicion, and directions had been given that it should be boiled before being used for drinking purposes. Patient had returned from Folkestone to town at the end of October, and had been in her usual health, except for slight headache during a fortnight, until November 23rd, when she was taken ill with shivering, frontal headache, pains in back and limbs, giddiness, nausea, and cough without expectoration. The shivering was repeated several times in the four days' interval between date of attack and admission. On the fourth day of illness, the skin was hot and dry, the lips parched, cracked, the tongue coated, and bowels confined. No physical signs could be discovered except some splenic enlargement. On the 8th day, imperfect dullness and patchy crepitations were observed at the left base, with bronchial breathing. On the 10th day, some tubular breath-sound was also heard at left sub-clavicular region. On 13th day, there was patchy dullness and crepitation over the *left* back and front of the chest. Exaggerated breathing in *right* sub-clavicular region, but crepitation also scattered over the axillary region and posterior base. Rusty sputa. Delirium.

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[1] Clinical Lecture on a case of Acute Pneumonia. Medical Times and Gazette, Feb. 25, 1860.

On the 14th day, the note was as follows:—*Right side,* considerable tubular resonance over 10th and 11th ribs posteriorly: dullness above, to include scapula. Coarse râles at extreme base, inspiratory crepitation over scapula. In front exaggerated respiration; in lower axilla sub-crepitant râles. *Left side,* posteriorly, patchy dullness, coarse, scattered liquid râles. Anteriorly, dull in sub-clavicular region, with moist râles and some larger clicks. One doubtful spot suggestive of typhoid fever was observed on the abdomen. Death occurred on the morning of 16th day. The appended chart, Fig. 17, gives the temperature of this case and illustrates fairly well the more prolonged and fluctuating course of the fever, which characterizes typical cases of this variety from the more ordinary form of pneumonia.

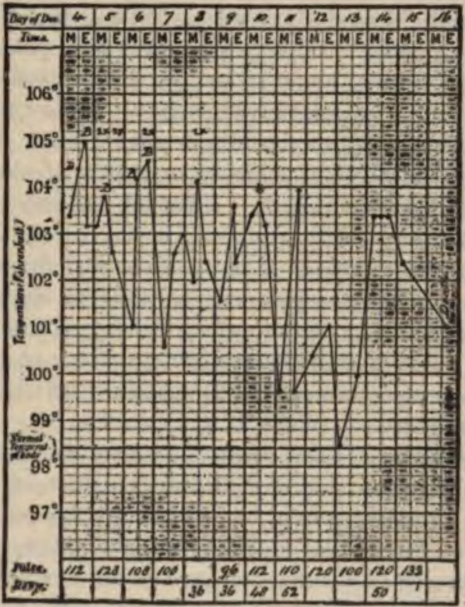

FIG. 17.—Illustrates case of pythogenic pneumonia. B = Cool bath. Qx = Quinine, 10 grs. * = Salicylate of soda, 20 grs. every four hours.

Post mortem, no lesions of enteric fever present. The upper part of the right lung was solid, except the extreme apex and anterior edge. Several wedge-shaped, dark patches were seen on section (recent hemorrhagic infarcts). In the neighborhood of these infarcts there were thrombi in the vessels. This was the case with the infarcts in the other lobes; the general appearance of the section was that of red hepatization, the central portions of a lighter color than the rest, and inclining to grey.

Right middle lobe. Soft and crepitant.

Right lower lobe. Quite solid, in appearance similar to the upper, also containing some infarcts.

Left upper lobe. Same as right upper lobe as regards area of consolidation. Color rather dark. Considerable œdema with some infarcts.

Left lower lobe. Fairly crepitant, except a patch of consolidation at its upper, and one at its lower edge.

Heart. Ante and post-mortem coagula in cavities of both sides. Muscular tissue firm.

The diagnosis in this case was at first very uncertain, and even when pulmonary signs made their appearance, they were of that scattered description not incompatible with their being a part of the manifestations of enteric fever. The enlargement of the spleen, again, pointed in the same direction, and although there were, with one doubtful exception, no spots present, one or two relaxed pale motions were passed. The extent of the pulmonary lesions, however, led to the diagnosis of typhoid or pythogenic pneumonia, which proved correct. Unfortunately no search was made for micrococci either before or after death. The character of the lesions described, however, is highly suggestive of their septic origin.

TERMINATIONS OF PNEUMONIA.—Although as a rule the consolidation of pneumonia clears up pretty uniformly in the inverse order of its formation, it certainly does not do so in all cases, and detached islets of resolving exudation sometimes give rise to physical signs—largish clicks and circumscribed blowing sounds, which it may be almost impossible to distinguish from those of pulmonary disintegration. A careful examination of the sputa in the manner already explained will help us much to a right view of such cases.

As already stated, the third stage of pneumonia, that of grey hepatization, may be altered for that of *diffuse suppuration* of the lung. The symptoms which attend this fatal change are of a typhoid type, the fever continuing shows a fluctuating range, the tongue becomes dry, brown and tremulous, the prostration is marked and attended with muttering delirium, sweating and sudamina may be present. The redux crepitations normal to the period of disease are replaced by coarse liquid râles; the respirations become increasingly rapid, and attended with rattling; the pulse rapid, compressible. Death soon terminates the scene.

Abscess, i. e., circumscribed suppuration of the lung, is, like the preceding condition, of uncommon occurrence in pneumonia. The symptoms attending its formation may be very slight. Rigors followed by sweatings towards the latter period of the disease would suggest suppuration. It not infrequently happens, however, that the first sign which enables us to recognize pulmonary abscess as a complication of pneumonia, is its discharge through the bronchus, and the sudden expectoration of a large quantity of pus. This having occurred, cavernous breath-sound and gurgling râles may be heard over a portion of the consolidation where the breath-sounds were perhaps before suppressed.

Gangrene of the lung is a condition not of uncommon occurrence in the later stages of pneumonia in cachectic subjects, and particularly in the intemperate. It more frequently met with in apex pneumonia in adult persons. The symptom which characterizes the onset of gangrene is fœtor of breath. There may be no other sign of the complication present, for the sphacelus need not be large, and until it has been evacuated no appreciable cavity may exist. The disintegration of the sphacelus is, however, attended with darkened fœtid sputa, which, on microscopical examination, will be found to contain fragments of lung-tissue.

Acute pneumonia may terminate in the *chronic* form of the disease.

Œdema of the lung may long persist after pneumonia in persons past middle life; it remains not infrequently a permanent condition.

PROGNOSIS.—More than 24,000 persons die annually in England and Wales of pneumonia, a mortality sufficiently great to place the disease in the front rank of those dangerous to life. Advanced age and alcoholism are the most important states unfavorable to recovery from pneumonia; pre-existing chronic disease, *e. g.*, Bright's disease or diabetes, are likewise very unfavorable elements in prognosis. The severity of attack varies much in individual cases, and in the pneumonias prevalent in different years. Hence the *extent* and *type* of the disease must in each case be taken into account. It will be obvious that double pneumonia is much more serious than single. The prognosis is probably far more serious in the pythogenic forms of pneumonia, but such cases differ greatly, and we have no statistics by which their mortality can be exactly compared with that of ordinary pneumonia. The danger in these cases arises more from the general state than from extent of lung involved. In children pneumonia runs generally a favorable course, and the consolidation melts away sometimes with astonishing rapidity. *Apex pneumonia,* excluding cachectic (especially phthisical and alcoholic) cases is not more dangerous than basic pneumonia, and recovery may be quite as complete in the one as in the other. Pneumonia is a relatively fatal malady in those whose nervous systems have been "used up" by previous mental anxiety or overstrain. For an admirable discussion of the effects of different methods of treatment upon the mortality I must refer the reader to Dr. Sturges' work.[1] The two conclusions that prominently arise out of a careful consideration of the subject are: (1) That pneumonia will ever claim a certain proportion of victims, all our efforts to the contrary notwithstanding. (2) That above this certain small percentage of fatal cases, there is a highly fluctuating margin, in which elevation or depression of the death-rate is greatly dependent upon treatment. That we have not yet arrived at the lowest possible mortality from pneumonia the most recent statistics, if we may trust them, show.

Of 1,065 cases collected by the Investigation Committee, the mortality was about 1 in 5½, and subtracting from the gross total the intemperate, the mentally or physically depressed, the infectious cases, and those occurring in aged people (above 65 years)—withdrawing, that is to say, all the principal causes of fatality—the death-rate is still 1 in 8:[1] a rate not greatly less than that of all cases admitted to five of our large London hospitals.[2]

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[1] On Pneumonia, 1884, p. 200.

The subjoined table gives a brief analysis of the cases, 42 in number, which have come under my own observation at the Middlesex Hospital, with an average of 30 beds, between July, 1880, and December, 1883.

Cases.	Age.		Death.	Remarks.
7	under	10	0	
13	between	10–20	1	Pythogenic case, see chart, p. 135.
17	"	20–40	5	Two intemperate, both complicated with delirium tremens, one of them dying on forty-eighth day of gangrene of the lung. One case complicated with heart disease.
3	"	40–60	0	
2	"	60–80	1	Aged 75.
42			7	

Of the 42 cases 7 died, of which 5 would come under the category of special causes, viz., advanced age, intemperance, and blood poisoning.

Exceptional symptoms in pneumonia and their bearing on prognosis.—In children and very rarely in adults of unstable, nervous systems, *convulsions* may replace rigors at the onset of pneumonia. *Jaundice* is not infrequently observed in slight degree in the early stages: it is sometimes well-marked. *Delirium* is occasionally a marked feature of the disease, and is always a grave symptom. In intemperate subjects delirium tremens is very apt to complicate pneumonia. The *temperature* sometimes mounts above 105°, also in cases of great gravity.

Decided *hæmoptysis* is rare in pneumonia, and is regarded by Walshe as significant of tubercle. I have seen several examples, however, in which rather sharp hæmoptysis has attended pneumonia, which has nevertheless run otherwise the usual favorable course.

A brick-dust rustiness of sputa is the usual degree of sanguineous tinge, in the first days of pneumonia: a darker and more *prune juice color* is, however, sometimes observed in old people, and in them it is not necessarily of unfavorable augury. The absence of any coloration of sputa, in otherwise well-marked pneumonia, would lead me, from experience, to fear a tardy or incomplete convalescence. I have observed two well-marked examples of dense consolidation of lung from pneumonia, involving the whole lung, in which breath-sound was inaudible, and vocal fremitus correspondingly diminished. The diagnosis was arrived at principally from the very slight cardiac displacement being quite inadequate, as the result of so extended an effusion into the pleura. In both cases redux crepitation was subsequently developed, and the consolidation cleared up. Doubtless the absent breath-sound was due to an unusually large overflow of exudation into the small bronchi.[1]

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[1] Collective Investigation Record, vol. ii. Report on Acute Pneumonia, p. 66.

[2] Loc. cit., p. 51.

TREATMENT.—Before speaking of treatment it is important again to observe, that pneumonia differs greatly with regard to severity and type in different epidemics, and that the subjects of pneumonia are individuals differing, perhaps even widely, in constitution and powers of resistance. Hence, while there may be a general plan of treatment best adapted to steer the patient through the dangers of this disease, there is room for much diversity in detail, and no one system of treatment can be accepted for all cases.

The difficulties and dangers which arise in the course of the disease, are grouped about its four consecutive periods of *hyperæmia, consolidation, crisis* and *resolution.*

Absolute confinement to bed in a good, airy room, warmed to a temperature between 62° and 65°, with the air slightly moistened, and a careful, obedient nurse, are of the first importance in the treatment of all cases of pneumonia.

(1) *Stage of hyperæmia.*—In the first stage of the disease (*a*) shock; (*b*) pyrexia; (*c*) pulmonary congestion; (*d*) pain; chiefly attract our attention with regard to treatment.

The *shock* at the commencement of the disease, immediately after the rigor, is often considerable, especially in old people and young children, although it is rarely so marked as in acute bronchitis. Alcoholic stimulants may be needed at this period, but quietude in bed and nutritious soup are the best restoratives; a few doses of bromide with aromatic ammonia may be required.

We can frequently only conjecture the cause of the *pyrexia* in the early hours of pneumonia, and considering the nature and peculiar dangers in the later stages of pneumonia, perhaps the worst treatment of this symptom is that very generally adopted, viz., the administration of aconite. The favorable issue of the disease usually turns upon the maintenance of heart power and vessel tonicity, both of which are lowered by aconite at the very outset, sometimes beyond recall, as has happened in cases that have come within my experience.

The pyrexia of pneumonia is of brief duration, and if the temperature, which should in all cases be carefully watched and recorded, do not mount above 104°, it *per se* requires no energetic interference. Tepid sponging will slightly moderate it and be comforting to the patient; cool drinks and saline medicines will be similarly useful. If the temperature mount to 105°, five-grain doses of quinine, repeated every three hours, with perhaps a ten-grain dose to begin with, should be given to the adult. In children, who can be lifted about with great ease, the temperature may be kept under by the warm bath at 90°, employed as often as may be necessary, with smaller doses of quinine. In adults this measure is, in private practice, more difficult and disturbing to carry out.

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[1] Dr. Petrone, Lo Sperimentale, Nov., 1882, records six cases of what he specifies as solid pneumonia; one occurring in his own practice, and the others related by MM. Grancher, Brissaud, Beurmann and Leroux, in which the dullness, absence of fremitus, and breath-sounds resembled the signs of effusion, and were accounted for post mortem by fibrinous exudation extending into the small tubes.

Supposing the temperature, however, to show a tendency to rise in spite of quinine and sponging, the cold pack or the cool bath, *i.e.*, at 80° or 70°, must be had recourse to. Ice bags to the head or Leiter's tubes are very useful in adults in aid of anti-pyretic measures. Kairin is too prostrating a drug for employment in the early stages of pneumonia. Anti-pyrin is less so, but I cannot from present experience confidently recommend it.

In this pyrexial stage of pneumonia the bowels should be well cleared by a few grains of calomel, and a saline aperient. Liquor ammoniæ acetatis, and citrate of potash, are most useful remedies, having, with the help of a saline purge, the effect of relieving blood-pressure by natural elimination from skin, kidneys, and bowels, instead of emptying the arterial into the venous system, as is the effect of drugs of the aconite class when given in efficient doses.

Rest and saline medicines diminish hyperæmia, and the measures for the relief of pain have the same tendency.

In old people and children poultices are sometimes found to be oppressive, and will then be better exchanged for cotton-wool applications covered with oiled silk. It is very important in all cases to avoid hampering the movements of the sound side of the chest.

The *pain* of pneumonia, which indicates the seat of lesion and aggravates dyspnœa, should be treated by local rather than general measures. Opium, or the subcutaneous use of morphia, may be necessary in exceptional cases, but they are not desirable means of combating this symptom. Hot poultices frequently renewed, due care being taken to apply them with as little disturbance of the patient as possible, are valuable in pneumonia in relieving pain by lessening arterial tension in the pleura. This they effect by dilating the superficial capillary distribution of the intercostal vessels concerned. When the pain is severe, from four to a dozen leeches may be employed with great relief, and it is sometimes well to encourage the bleeding by the immediate renewal of poultices or fomentations. The condition of the patient, and especially the evidence of venous plethora under existing circumstances, *e.g*, amount of lividity and hardness of pulse, will guide us with regard to the number of leeches. In country or colonial practice cases may very possibly be met with in which a venesection to eight or twelve ounces may be most useful at the first onset of pneumonia. I have not met with cases as yet in which more than local depletion has been indicated, but from local depletion I have often seen good results. In cases of severe pain, where leeches are not desirable, a blister 3×3 applied underneath the poultice is often valuable. Cold applications are recommended by many physicians; I cannot say that I have been at all impressed with their utility. As a rule, stimulants are not needed in the treatment of this stage of pneumonia, but in cases of typhoid type with high fever, delirium, tremulous tongue, and rapid, compressible pulse, they must be had recourse to in sufficient and properly regulated doses, the alcohol being given alternately with a mixture containing carbonate of ammonia. The habits of the patient must be borne in mind in estimating the quantity of stimulants required. In cases of low type, in which the features of the disease remind one of idiopathic erysipelas, it may be good practice to place the patient at once on 20 or 30 minim doses of tincture of iron, with half-ounce doses of liquor ammoniæ acetatis, and this plan in alcoholic cases will sometimes answer without the aid of alcohol.

The diet of the patient must consist of nutritious fluids, milk, strong beef-tea, mutton or chicken broths, with perhaps some farinaceous thickening. The milk may be diluted with effervescing water or flavored with tea or coffee. Cream is sometimes a useful addition to the dietary. The food and stimulants should be given at reasonable intervals of about two hours, and in reasonable quantity, adapted to the exigencies of the case. It has occurred to me sometimes to see a patient overdone with food, his circulation overloaded, and the abdomen distended from many pints of fluid, much of which is waste, at a time when the respiratory functions are almost in abeyance, the right heart is already embarrassed, and elimination of unused material is difficult.

(2) *Stage of consolidation.*—The next period for treatment is that of consolidation of the lung. Continued pyrexia and pain, with still extending disease, and possible invasion of the opposite lung, attend this period. And thus far, the treatment is but a continuance of that already laid down.

At this period of the disease, from the 4th or 5th day to the crisis, life is most usually threatened in severe cases by, (1), failure of heart; (2) hyperæmia (not truly inflammatory, but as a consequence of heart failure and vessel paralysis) of the sound lung.

In severe cases stimulants are necessary, and they may be often usefully combined with quinine. Two to five grains of quinine should be given in milk, so that the patient gets from ten to twenty grains in the twenty-four hours, and as much brandy or wine as his symptoms demand. If symptoms of failure of heart be observed, and often in anticipation of such arising, small doses of digitalis (five to ten drops of the tincture) should be added, either to the stimulant or to the mixture.

It is sometimes advisable to substitute for the salines ammonia and bark, in other cases iron and acetate of ammonia. Excessive pyrexial symptoms must be met as before. With regard to the usefulness of moderate doses of digitalis in this period of pneumonia, whilst speaking from convincing experience of the fact, I would point out that this use of the drug is consistent with the most rational aim in the treatment of this disease. Salines, poultices, leeches, and possibly venesection, have the common object of lessening pulmonary congestion, easing arterial tension, and depleting the *venous side* of the circulation; supporting foods, and, when necessary, alcohol, quinine and digitalis, again pull together in maintaining heart power and tonicity of vessels at the period when these tend to fail. There are many cases of pneumonia, however, in which the simplest possible treatment with the fewest drugs is the best.

It is a small detail to notice, but one by which much comfort may be given to the patient, especially at this stage, when the respiratory passages are dry, hot, and often sore, viz., to direct the nurse to anoint the aperture of the nostril with a little sweet oil, which, by trickling into the passages, keeps them lubricated.

At the stage of the disease now under consideration the symptoms and signs reach their acme, and the strength of the patient its ebb: the crisis is anxiously waited for, and is not unattended with dangers of its own.

(3) *Period of crisis.*—The rapid fall of temperature that occurs where *crisis* is well marked in pneumonia, and the copious sweating or other phenomena attending it, are frequently associated with considerable shock and exhaustion. Failure of heart and pulmonary œdema are the special sources of danger. These must be met by carefully supporting the patient, temporarily increasing the stimulants to any necessary amount, and by giving moderate, three grain, doses of quinine, in combination, perhaps, with small doses of digitalis. Port wine may often at this period be substituted for brandy.

The dangerous symptoms associated with crisis pass in a few hours, although sometimes for a day or two the temperature remains sub-normal. At the termination of crisis it is often wise to suspend medicinal treatment altogether for a time, continuing the poultices, and regulating the diet to longer intervals. Until critical discharges have ceased, no important alteration should be made in the dietary. No active measures should be taken to arrest such discharges.

(4) *Resolution stage.*—During the early part at least of this fourth period of pneumonia (as we have artificially divided the phases of that disease), the same recuperative measures should be continued, strict rest in bed being still enjoined, the dietary being gradually improved, some solids allowed, and the stimulants (if any used) cautiously curtailed, and changed to port wine or malt liquors. The condition of the tongue will be the guide on these points.

The kidney and bowel secretions must be looked to, for it must be borne in mind that inflammatory products are being absorbed and eliminated. After a few days, simple quinine or quinine and iron tonics, or a little mineral acid twice daily, may be usefully employed. The resolution as a rule proceeds steadily and satisfactorily. In some cases, however, as already pointed out, a secondary and recurrent rise of temperature, with slight chills and sweatings, attend the resolution process; quinine must then be steadily côntinued, or if this drug disagree, then arsenic may be tried, the patient being very strictly kept at rest.

After a consolidation has cleared up it is not uncommon for there to be some return of crepitation over the seat of past pneumonia. This, which is doubtless due to local atonicity of vessels favoring a passive congestion, is best treated by the employment of tincture of iron internally. A very analogous condition is not infrequently met with in acute nephritis, in the course of convalescence from which we may get increased albumin, and some return of blood in the urine, without any associated rise of temperature.

Counter-irritants are occasionally useful in resolving pneumonia, and two or three grains of iodide of potassium, in combination with citrate of iron and quinine, will sometimes hasten convalescence in indolent cases.

Change of air is needed to complete convalescence after pneumonia, and in no disease is it more important that convalescence should be complete. The only conditions that need be specially observed with regard to such change, are the avoidance of damp, low-lying, ill-drained localities, and the choice of places where the patient can get out on level ground. Lymphatic and scrofulous persons will recover best at the seaside, the nervous and bilious subjects inland.

Sphacelus of the Lung.—In rare instances it happens that the expectoration in pneumonia becomes fœtid, or distinctly gangrenous. In slight cases such symptoms will yield to the frequent use of respirators charged with eucalyptus, ten drops with an equal part of spirit to be inhaled for half an hour every two or three hours, or Robson's dry eucalyptus or pine-oil spray may be kept in more or less constant use about the bed, the patient occasionally inhaling the spray more directly.

When the sloughing of the lung is extensive, and a gangrenous cavity is formed, it may be necessary to call in surgical assistance. (See Chap. XV.)

In *purulent infiltration* of the lung we cannot do more than try to support the patient by bark and ammonia, wine and food.

In *circumscribed abscess* we must endeavor to keep the abscess cavity as empty and disinfected as possible; helping expectoration by change of posture, employing disinfectant inhalations as for sphacelus, and keeping up the patient's strength. Surgical interference may again in these cases be necessary, but it should not be too hastily had recourse to, the abscess frequently contracting under ordinary treatment.

CHAPTER XI.

BRONCHO-PNEUMONIA.

BRONCHO-PNEUMONIA or lobular inflammation of the lung is an affection partly exudative, partly catarrhal, of certain lobules of the lung and of their associated bronchioles. The inflammatory lesion is, however, not always strictly confined to separate lobules or groups of lobules, for in some cases and especially in those originating in blood conditions, more diffused areas of lung are affected, being less restricted by lobular divisions; in other cases again the inflamed patches are not terminal to bronchi but extend laterally from them.

ETIOLOGY. — Broncho-pneumonia is most commonly met with as a primary disease in infancy and childhood. It may occur at any age as a disease secondary to capillary bronchitis, whooping-cough, measles, tracheotomy, diphtheria, pyæmia and allied conditions, and phthisis, but is nevertheless most common in childhood.

The mode of origin of the disease may be:—(1) By extension from bronchitis. (2) By congestion and stasis consequent upon lobular collapse of lung, a condition of frequent occurrence in children with weak walled or rickety chests, in the course of bronchitis and whooping-cough. After tracheotomy, when the mechanism of cough or rather of expectoration is spoiled, lobular collapse and consequent pneumonia is very common. (3) By inhalation of inflammatory or septic products. In capillary bronchitis the inhalation of the bronchial products into the alveoli will set up alveolitis. In cases of tracheotomy again, there is a tendency for changed blood, purulent secretions and septic matters to become inhaled into the lungs and to set up lobular pneumonia. In diphtheria, during the period of softening of the membranes, there is, apart from tracheotomy, a disposition to septic involvement of the lobuli. In phthisis the inhalation of blood or sputa in course of expectoration frequently sets up new centres of lobular inflammation. The inhalation of dust or acrid vapors may possibly cause broncho-pneumonia. (4) By conveyance of morbific septic agencies through the vessels as in pyæmia, septicæmia and tuberculosis. Here the lesions, as before said, may not be strictly lobular, but affect vascular rather than bronchial territories. (5) By extension from deposits in the lung, chiefly tubercular.

SYMPTOMATOLOGY.—In describing the clinical features of broncho-pneumonia, cases of infantile form such as those arising primarily, or in association with bronchitis or whooping-cough, will be especially in mind.

lobe are affected, and by their juxtaposition the lung is more or less densely consolidated.

Confluent broncho-pneumonia may be associated with ordinary bronchitis of catarrhal origin, and very often arises in the course of whooping-cough. It comparatively rarely arises from the other causes of broncho-pneumonia. The symptoms of the disease are identical with those of the preceding variety, except that pleuritic pain is more commonly experienced on the affected side.

The physical signs are somewhat different. The pulse and respirations are similarly quickened, and the signs of obstructed inspiration are observed but not symmetrically, the deficient expansion on one side being, in cases where the disease is at all extensive, notably greater than on the other. Most commonly the posterior and lower portion of one lung is affected, and over this region the percussion note is distinctly impaired, in the earlier stages having a somewhat tympanitic quality, but as the lobules coalesce becoming more toneless. Vocal fremitus in young children is of no value, but on auscultation the cry of the child is bronchophonic in character with a tendency toward ægophony. Children, however, who are seriously ill with broncho-pneumonia rarely cry. The breath-sound over the consolidated area is weak and bronchial, being considerably masked by abundant sub-crepitant râles of very sharply defined or metallic character. The disease may affect both sides, but as a rule the oppcsite lung is affected with bronchitis only, or it may be with a few centres of disseminated pneumonia.

The temperature and other phenomena in this form of the disease are the same as in the preceding. There is a greater tendency for this form of broncho-pneumonia to become chronic and to run a long course of perhaps many months, terminating in pulmonary fibrosis with bronchiectasis or in a form of phthisis.

In other cases the disease proceeds immediately to suppurative destruction of lung and to the death of the patient, and in yet other cases the mere extent of the disease in the early stage proves fatal.

The signs of suppurative destruction are, increased adynamia, maintained rapidity of pulse, fluctuating temperature, and hectic sweatings with rapid loss of flesh. The râles become larger, more bubbling, even gurgling in character, and although children rarely expectorate, it is obvious that much secretion comes up to the throat, and irritative diarrhœa with slimy stools frequently supervene.

The prognosis in the confluent form of broncho-pneumonia is, however, in a given number of cases more favòrable than in the disseminated form. The bronchitis of the opposite lung may clear up, and time is thus allowed for reparative changes to take place in the affected lung. These changes sometimes go on with great rapidity, but never so quickly as may occasionally be observed in the lobar pneumonia of children.

TREATMENT OF BRONCHO-PNEUMONIA.—The treatment of broncho-pneumonia is mainly that of bronchitis with careful support of the patient. In the disseminated form of the disease it is doubtful if poultices are of any service, and it is most important that the respiratory movements be allowed as free play as possible. In the more localized confluent form of the disease poulticing is sometimes useful. The timely use of emetics in whooping-cough will sometimes avert the occurrence of broncho-pneumonia, but it must be allowed that the disease is often started by the imprudent exposure of children with whooping-cough to the external air, under the delusion that in this disease such exposure is harmless or beneficial. A careful nurse or mother who understands how to hold a child during the paroxysms of whooping cough, so as to permit the fullest play to the respiratory muscles, may help much in averting pulmonary collapse and subsequent pneumonia.

The room should be kept at a temperature between 62° and 65°, the air moistened, and in some cases the addition of a little tar water to the bronchitis kettle is useful. The temperature of the patient if high can be kept within bounds by the occasional use of the warm bath 90° to 95°, and the head may be sponged with water, cooler than this but not cold.

The secretions must be kept clear, the patient well supported by beef tea, chicken broth, veal tea, milk, cream, and when stimulants seem requisite they should be liberally given. In all septic cases they are required in full doses. Port wine is an excellent stimulant, and may be combined with a few minims of Battley's cinchona, or with small doses of quinine; or brandy may be added to the milk or broth.

Careful support of the patient and good nursing are of infinitely more importance than medicine, but sometimes a little ammonia and ipecacuanha seem useful.

In the convalescent stage of both varieties of this disease, the practitioner must bear in mind the frequent presence of rickets, of a delicate family history, and the decided tendency to remnants of disease being left behind in the lungs or bronchial glands. Cod-liver oil with steel wine, containing a few minims of syrup of the iodide of iron, are now valuable, and a change of air, to a warm seaside place if possible, is very important.

CHAPTER XII.

NARROWING AND DILATATION OF THE BRONCHI.

NARROWING OF THE BRONCHI.—General narrowing of the bronchi is practically only met with as a consequence of swelling of mucous membrane in catarrhal affections, or of exudation in croupous and diphtheritic maladies. Doubtless cases may occur in which there is some general diminution in the calibre of the bronchial system, but they are not recognizable during life. In association with atrophic and contractile changes in the lung, whether congenital as atelectasis, or from disease, the tubes traversing the affected part are widened, never contracted.

Localized narrowing of a bronchus may arise:—1. From cicatricial contraction of an ulcerated surface within the bronchus. 2. From contractile sclerosis of the bronchial sheath at one or more points. 3. From invasion of the calibre of the bronchus by malignant growths. 4. From pressure upon the bronchus by enlarged glands, growths, hydatid, or aneurismal tumor.

1. The cicatricial changes ensuing upon ulceration are amongst the very rare cases of bronchial narrowing and are always of syphilitic origin.

2. In association with the more chronic indurative form of phthisis, it is not uncommon to find bronchial tubes narrowed or even obliterated by what may be regarded as cicatricial growths involving the sheath of the bronchus, (*a*) at points adjacent to tuberculo-fibroid nodules: (*b*) at the entrance to cavities which have undergone considerable or complete contraction, and are surrounded by a zone of cicatricial induration in which the entering bronchus is involved. In this way partially contracted cavities are not infrequently closed.

3 and 4. The invasion of malignant growths and the pressure of tumors are amongst the most common causes of obstruction to the main bronchi; and are of too obvious a mechanism to require further exposition.

The necessary consequence of narrowing of a bronchus at any point is more or less complete retention of secretion behind, and changes of a destructive but variable kind in the lung. Sometimes a cavity becomes thus shut off by occlusion of its communicating bronchus. Its purulent contents may then collect, and subsequently inspissate into a creamy *débris* which may at a later period become cretaceous. In other cases the purulent secretions increase, become pent up, causing elevation of temperature and other signs of abscess, and finally when they attain to a certain degree of pressure the narrowed bronchus yields, and a discharge takes place, after which accumulation again commences.

In cases, however, in which the secretion is derived from the bronchial mucous membrane, the lung not being primarily involved, it is thick, viscid, and more purulent, and collects in such quantities as to distend the bronchi behind the obstruction. When sufficient distension has taken place a paroxysmal cough will expel through the narrowed orifice a certain portion, the overflow so to speak, of the collection, in the form of thick viscid and more or less nummulated sputa, generally not offensive.

The condition of lung that attends narrowing of a bronchus is in the later stages one of airless collapse. Dr. Pearson Irvine contended in an able series of papers in the Pathological Transactions, for 1877 and 1878, that emphysema was the primary consequence of bronchial pressure, since the effect of the narrowing would be to impede expiration, whilst inspiratory effort tending to widen the narrowing would be successful. It is obvious, however, that so soon as secretion begins to collect behind the obstruction, the air cells can no longer be penetrated by air, whilst that which remains in them must slowly be expelled. Secondary changes of a destructive character not infrequently ensue in a lung whose main bronchus is occluded by pressure, changes which are regarded by many as arising from disturbed innervation through pressure upon the pulmonary plexuses,[1] but although this pathology may hold good in some cases, it is probable that as a rule the pulmonary destruction is the result of the retention of pent-up secretions, and is analogous in origin to similar conditions of kidney ensuing upon hydro or pyo-nephrosis.[2]

The *symptoms* and *signs* of bronchial narrowing need only be considered with regard to those cases in which the constriction is situated at one or other main bronchus. In these cases the signs of narrowing of the bronchus sometimes present themselves, before the cause of that narrowing can be precisely made out.

Relatively feeble breath-sound of harsh or blowing quality over one lung, and particularly over the upper interscapular region of the affected side are the first signs to be observed. The percussion note, at first unaltered, becomes of higher tone, the vocal fremitus and resonance being lessened on the affected side. The more or less exaggerated vesicular breath-sound over the unaffected side, is in marked contrast with the feebleness of breathing, and want of vascularity on the affected side.

As the case proceeds, the feebleness of the breath-sound becomes increased to final extinction; low-toned sonorous rhonchus is heard throughout the affected side, and with increasing loudness and roughness as the interscapular region is approached. Impairment in the mobility of the affected side comes to be observed, then most usually some shrinking of the side and percussion dullness over the affected lung.

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[1] Sir William Gull, Guy's Hospital Reports, 3rd series, vol. v., p. 309, 1859.

[2] See article on Mediastinal Tumors by the Author, Reynolds' System of Medicine, vol, v., in which this matter is more fully discussed.

From the first there is cough, which may be of laryngeal type from involvement of the nerves of the larynx, coincidently with the bronchial compression. The cough is, however, generally spasmodic in type, the passage of the thick sputa through the stricture giving rise always to more or less, sometimes to very severe, paroxysms of dyspnœa. As pulmonary collapse proceeds, pleuritic pains and the signs of dry pleurisy present themselves, and, with advancing dullness, may lead the observer away from the right diagnosis. The heart is uncovered and shifted towards the affected side, unless its position be otherwise determined by the pressure of a tumor.

In the most common class of cases, those in which the narrowing is due to the pressure of an aneurism or malignant growth, the signs of tumor will be developed at an early stage of the disease.

In cases of syphilitic narrowing there will be the history of syphilis, the signs of narrowing and the absence of those of tumor. In an interesting case that has recently come under my notice, in which the left bronchus was almost obliterated, and the lower portion of the trachea narrowed by syphilitic cicatrices, in addition to an absence of the signs of aneurism, it was observed that the patient's distress was greatly increased by an attempt to lie in the prone position, and that he felt most easy when erect and walking about the room. In aneurism the patient's troubles are much increased by movement.

From the profuseness of the expectoration in the later stages of bronchial narrowing in association with consolidation of the lung, these cases are sometimes mistaken for phthisis, at other times for pleuro-pneumonia or empyema. A careful consideration of all the signs present will be necessary to establish the diagnosis, for these are undoubtedly among the most difficult cases in chest diagnosis.

TREATMENT.—Much may be done in the way of giving ease in most cases. The cause of the constriction, be it aneurism or syphilis, must be appropriately treated.

Small, regulated doses of chloroform for inhalation are valuable, in giving relief to spasmodic cough and in aiding expectoration. In cases in which paroxysmal dyspnœa arises from compression of the bronchus by aneurism, nitro-glycerine (1 minim of 1 p.c. solution) will give relief by lessening tension within the sac. Expectorant remedies are worse than useless. Chloral and paraldehyde give more promise for usefulness than opiates.

When the main bronchus is involved the prognosis is of the gravest character.

DILATATION OF THE BRONCHI—BRONCHI-ECTASIS.—Chronic bronchitis and emphysema commonly entail some general enlargement of the bronchial tubes, the widening being most apparent in the smaller divisions of the tubes. At portions of the lungs, as the apices, which are but imperfectly supported externally, the bronchial dilatation may be so considerable, and attended with such changes in the surrounding tissue, as to merit the term bronchi-ectasis.

Bronchi-ectasis consists of a manifest widening of a more or less limited portion of the bronchial tubes. There are two forms of bronchi-ectasis, the *cylindrical* and the *sacculated.*

In cylindrical or fusiform bronchi-ectasis, the dilatation involves some length of the tubes, varying from a few inches to a system of tubes ramifying through an entire lobe. The enlargement is uniform throughout the length of tubes affected. In sacculated bronchi-ectasis a restricted portion of a tube is enlarged to a globular form, from half an inch to an inch in diameter. The whole calibre of the tube is as a rule involved, and the ectasia may be solitary or there may be many scattered through the lung. As a rule this form of bronchi-ectasis is situated at the peripheral portions of the lung, corresponding with the smaller bronchi, and small openings lead from the rounded and apparently closed distal side of the sacs, to fine tubes, the branchlets of the widened bronchus.

Bronchi-ectasis is never a primary disease, but is dependent upon either increased pressure within the tubes, or traction upon their walls.

The following may be enumerated as the causes of bronchi-ectasis:—

1. Increased air-pressure during cough, acting principally at those portions of the lungs where there is least support, notably the apices. This cause becomes operative in the course of chronic bronchitis and emphysema, and assists the action of those causes which bring about damage to the texture of the bronchi.

2. Obstruction of a bronchus, whether from intrinsic cicatricial changes or growth from the lodgment of foreign bodies, or from the external pressure of an aneurism or other tumor (see Narrowing of the Bronchi, p. 148). The bronchial tubes behind the obstruction become distended by accumulation of mucous secretion, and the lung surrounding them condensed and airless. The dilatation of the tubes in this form of the disease is of the cylindrical kind, affecting most notably the tertiary branches, which are less firmly supported by cartilaginous plates, but the minutest bronchioles, and even the infundibula, become more or less occupied with muco-purulent secretion.

3. When the vesicular texture of a lung is obliterated by permanent collapse or contractile growth, the tendency of the inspiratory force is to widen the bronchial tubes traversing that lung; and any further contraction of the condensed tissue in which the tubes are imbedded will further widen them.

(*a*) Secondary to a pneumonia or a dry pleurisy, a portion of a lung, or perhaps a whole lobe, will in certain cases become condensed by connective tissue growth, extending to its interstices from the pleura along the interlobular septa, and from the sheaths of bronchi and vessels penetrating it. The result of such fibrous over-growth—which may be termed chronic interstitial pneumonia, leading to cirrhosis of the lung—is necessarily a contraction of the lung from collapse of its vesicular structure. The pleural surfaces of such a lung are invariably adherent. It is obvious that any inspiratory effort telling upon such a lung must tend to widen the elastic bronchial tubes with which it is penetrated.

(*b*) Such a lung being held attached at its circumference by the pleuritic adhesions, and its bronchial tubes branching to the periphery from the root, it is clear that when the thoracic wall has receded, and the surrounding organs, thoracic and abdominal, have approximated as far as possible, any further contraction of the lung must drag upon the bronchial tubes and extend their calibre in all directions. The medium and sub-capillary tubes imbedded in the depth of the lung are most affected, and become widened out into fusiform and globular cavities, the finest tubes become extensively obliterated in the course of the contractile disease. The secretions of the widened tubes tend to collect from the shape of the tubes, and the hardened tissue by which they are surrounded, rendering effectual expulsion with cough impossible; decomposition of the stagnant secretions follows, resulting in irritative inflammation of the cavities and adjacent parts. Neighboring bronchi-ectatic cavities may coalesce from atrophy and softening of their adjacent walls, and extensive irregular lobulated excavations of the lung may thus arise.

The bronchial cavities just spoken of often closely resemble true pulmonary vomicæ. The distinction is made *post mortem* by finding on the cavity walls remnants of bronchial membrane, with its characteristic columnar epithelium.

(*c*) In cases of thoracentesis for old standing empyemata, bronchi-ectasis amounting to complete loculation of lung may arise in a manner not essentially different from that just described. In such cases obliteration of the pleural cavity is finally affected from above downwards, by the gradual growth of adhesions agglutinating the approximated pleural surfaces.

The parenchyma of the lung is, however, in these cases, irretrievably condensed, so that the degree of enlargement of the organ necessary to fill the remaining thoracic space is only to be obtained by widening out the bronchial tubes, and further contraction of the fibrous lung must continue to have the same effect. Thus, the lung in some cases comes to be a mere shell enfolding bulbous bronchial cavities.

It is noticeable that in these cases, unlike those just considered, profuse and fœtid secretions rarely collect in the bronchial cavities. This difference arises from the fact that the thoracic space is mainly filled by the expansion of the upper part of the bronchial system, the base of the lung remaining collapsed. Hence drainage is better secured, and the cavities not being irritated by stagnant secretions, become comparatively dry and inert.

(*d*) In congenital collapse of any portion of the lungs, *atelectasis pulmonum*, the bronchial tubes ramifying through that portion are widened.

SYMPTOMATOLOGY.—It is obviously impossible to separate the symptoms and signs of bronchi-ectasis from those of the pulmonary lesions with which this condition of tubes is associated. The expectoration is profuse, muco-purulent, nummulated, with a tendency to diffluence, often very fœtid, and yielding on examination neither elastic tissue nor tubercle bacilli. It is expelled with violent paroxysmal cough, often terminating in vomiting.

In cases in which the bronchial dilatation is secondary to pressure or contraction of the bronchus, the expectoration is most viscid and mucoid, is not as a rule fœtid, and is expelled with considerable difficulty in successive thick, viscid masses at such times as there shall have been sufficient accumulation to force the narrowed passage. In these cases, as soon as the bronchi become distended with secretion, all breath-sound is extinguished, the affected portion of lung being at first imperfectly tympanitic, and latterly dull on percussion, and the affected side motionless and more or less retracted.

In bronchi-ectasis ensuing upon cirrhosis of the lung from any of the causes considered, we have together with the signs of such cirrhosis, profuse expectoration coming on at intervals with severe paroxysms of cough, the matter expelled being horribly fœtid, thin, and purulent, and often containing small curdy-looking masses: whiffs of fœtid gas precede and attend the expectorations. Yet in spite of these distressing symptoms the patient may, for a long time at least, suffer comparatively little in general health, nutrition and appetite being fairly maintained, and hectic but little marked. The physical signs vary according as the bronchi are filled or empty. After a severe cough with profuse outflow of sputa, diffused tubular breath-sound with mucous rattles may be heard over the affected side, to be obscured later on with fresh accumulation; dullness of percussion and more or less retraction of side are proper to the cirrhosis.

In cases in which neighboring bronchiectasis have broken into one another, a considerable area of excavation may be produced, usually in the supra-scapular region, over which cavernous breath-sound and gurgling râles with pectoriloquy may be distinctly heard, and the percussion note may have a perceptibly tubular character. In such cases the symptoms are more severe, hectic is more marked, nutrition suffers, and diarrhœa with red tongue are commonly observed. Elastic tissue is to be found in the sputa, but not the bacilli of tubercle.

The diagnosis is often extremely difficult between bronchi-ectasis and localized empyema, and especially so where the bronchi-ectasis is secondary to partial or complete occulsion of a bronchus.

Treatment.—In compression of the bronchus by aneurism or growth we can effect little by treatment. We may relieve whatever spasm there may be in association with the organic stricture, by small regulated inhalations of chloroform, a few drops at a time put on absorbent wool in a small bottle to be sniffed. I have known a pungent vapor, *e.g.*, of strong peppermint essence in hot water, sniffed through the nostrils, give relief to the spasmodic cough and aid expectoration. In aneurismal and syphilitic cases, large doses of iodide may be given. In fœtid bronchiectasis we are often but too helpless in effectual treatment. Tar, creosote, carbolic acid and eucalyptus may be given internally, and used as inhalations.

In cases where the patient is getting exhausted, and in which any definite area of excavation can be defined, exploration should be made by cutting cautiously down to the pleura, inserting a fine exploring trochar, and if the cavity be struck introducing a drainage tube. If no definite cavity be struck, it is well to insert the fine trochar in several directions, with the hope of setting up adhesive or cicatricial inflammation along its tracks. A very guarded opinion as to the ultimate good that will be effected by these measures, and their tentative nature, must in all cases be given, however, to friends.

CHAPTER XIII.

ABSCESS AND GANGRENE OF THE LUNG.

CIRCUMSCRIBED suppuration of the pulmonary tissues may arise from a variety of causes, the most important of which are, acute primary inflammation, wounds of the thorax, lodgment of foreign bodies, acute pneumonia in broken constitutions, circumscribed gangrene, abscesses secondary to pyæmia, and infective emboli.

Abscesses of primary origin are usually single. The most frequent causes of multiple abscesses are infective emboli and pyæmia. The collections of pus may vary in size from a pin's-head to that of a walnut. Those which form in association with acute pneumonia are of small size, numerous and irregular in shape. Abscesses occurring in connection with lobular pneumonia involve the terminations of the bronchi and assume a dendritic form.

SYMPTOMS.—The formation of pus in the lungs as in other organs, is usually attended with severe rigors and pyrexia, and in broken or debilitated constitutions often leads to great prostration. The first evident symptom of an abscess may be the sudden discharge of pus with fragments of lung tissue.

PHYSICAL SIGNS.—If the abscess be of any size the signs would be dullness on percussion, with gurgling râles and the other signs of cavity.

TREATMENT.—The treatment of abscess of the lung consists generally in the pursuance of the remedies for the diseases that may have led up to it. Quinine or bark and mineral acids are useful. Disinfectant inhalations are also of service, and the patient should be encouraged to evacuate the contents of the abscess cavity from time to time. Under these ordinary measures of treatment with good ventilation, and change of air at the earliest moment, abscess of the lung not infrequently heals. In some cases operative treatment is called for. (See Chapter XV.)

GANGRENE OF THE LUNG.

Death of a portion of the substance of the lung may occur under two forms; (*a*) circumscribed, (*b*) diffuse.

(*a*) The circumscribed form is that usually seen, the gangrenous area is distinctly defined, and may vary in size from that of a nut to a considerable patch involving the greater part of one lobe. The lower lobes and superficial parts of the lungs are those most frequently affected. The necrosed portions of the pulmonary tissue become moist, soft, pulpy, present a bluish-green color, and evolve a peculiar and highly offensive odor. The limits of the dead tissue are indicated by a zone of hyperæmia and consolidation. The dead tissue may slough and be discharged through a bronchus, leaving a ragged cavity behind. Occasionally the pleura is involved and the fœtid material finds its way into the pleural cavity, but this is rare, as adhesions usually form between the pulmonary and parietal layers of the pleura.

(*b*) In the diffuse form there is no line of demarcation between the dead and healthy tissue, but inflamed, congested and gangrenous lung tissue are all mixed up together. The greater portion of one lobe, or of an entire lung, may be affected, or perhaps both lungs may be affected at several points.

ETIOLOGY.—Gangrene may arise as a result of acute pneumonia, or from the inhalation of noxious gases. Pressure interfering with the circulation through the lungs, caused by mediastinal growths, and aneurism; foreign bodies in the air-passages or lodged in the lungs from without, *e.g.*, bullets, pieces of cloth, etc., may cause gangrene. It also occurs in drunkards and in the insane. It may complicate the asthenic fever left after such debilitating constitutional disturbances as small-pox, measles, typhus and the like. Pyæmic emboli, or the inhalation of fœtid discharges from cancer of the mouth or cancrum oris, or the bursting of a neighboring abscess into a bronchus, may lead to gangrene of the lung.

SYMPTOMS.—The most characteristic symptoms of gangrene of the lungs are the peculiarly offensive odor of the breath, associated with the expectoration of gangrenous material containing lung-tissue. The only condition likely to be mistaken for it, is old standing bronchi-ectasis suddenly becoming fœtid from necrosis of portions of mucous membrane, from which it is to be distinguished by the presence in the sputa of elastic fibres, and the previous history of the case. The general symptoms are usually those of extreme depression, asthenia, and collapse, death soon ending the scene. Hectic may be present in some cases.

PHYSICAL SIGNS.—In the early stages the auscultatory signs are indistinct and not to be relied upon, but later, if the gangrene be circumscribed, the physical signs are dullness over the affected area, with gurgling râles accompanied by an amphoric quality of breath-sound. The distinctness of physical signs, however, varies according to the degree of freedom of the cavity from the shreddy gangrenous tissue.

TREATMENT.—This should be nourishing and stimulating, alcohol being freely given. Antiseptic inhalations of creosote, carbolic acid or eucalyptus should be used. Antiseptics internally are of little use. In cases of circumscribed gangrene with marked hectic or septicæmic symptoms, and in which the locality of the gangrenous part can be fairly defined, paracentesis should be performed and an attempt made to drain the cavity; this proceeding is still further urged if there be reason to believe that the pleura is adherent and the surrounding lung consolidated. (See Chapter XV.)

CHAPTER XIV.

THE SURGICAL TREATMENT OF PULMONARY CAVITIES.

THE cavities of the lung in which artificial drainage may be sought to be effected by the aid of the surgeon, have their origin in:—

(1) Abscess of the lung, (*a*) Suppurative.
(*b*) Gangrenous.
(*c*) Hydatid.
(2) Bronchi-ectatic excavation of the lung.
(3) Tubercular or phthisical cavities.

There are certain principles common to the surgical treatment of all these forms of cavity, and there are certain precautions which should be borne in mind with regard to each.

The proper method of procedure in all—even the apparently most obvious and straightforward--cases is to divide the operation into two stages, viz:--1. That of exploratory puncture. 2. That for establishing drainage. In simpler words the surgeon must find the cavity before he can tap it, and present experience teaches that it is not easy to define a cavity with the stethoscope, and still more difficult to strike it with the trochar.

It must be said, too, that accurate auscultation with a view to strictly defining the outline of cavities for the purposes of surgery, requires a closer study on the part of physicians.

Distinction of pulmonary from pleural cavities.—It is often a very difficult matter to distinguish a pulmonary from a pleural cavity. The mistake of confounding an empyema with a lung cavity is chiefly of importance at the apices of the lungs, where it is most apt to be made, since there is less temptation to interfere with a lung cavity in this situation than in any other. Sometimes pleuritic cavities are here discovered *post mortem* which, had they been recognized during life, might have been dealt with and the patient saved. Having determined on operative treatment, however, the discovery that a supposed pulmonary cavity was really a local empyema, would be a matter for congratulation rather than otherwise. The clinical features most characteristic of a pulmonary cavity are as follows:—

Distinctness of breath-sounds, their hollow quality, and the presence of gurgling râles and cavernous splash on coughing. Whispering pectoriloquy is another essential sign of much importance in association with the other signs in distinguishing a pulmonary from a pleural cavity. It must be borne in mind, however, that one observation is often not sufficient for diagnosis, for if the cavity be filled with secretion, or its bronchus from any cause temporarily occluded, none of the signs may be obtained. In the case of cavities near the surface, a distinctly tubular percussion-note may be elicited, most clearly when the patient's mouth is open, contrasting with the dullness over a more or less extended area of the surrounding lung.

To define the outline of a cavity.—In investigating an area of excavation with a view to surgical treatment, a circle should be drawn to include the centre of greatest intensity of physical signs. Then using a stethoscope with a small chest piece, this centre should be gradually approached from all sides, and a mark made at each point where pectoriloquy and cavernous breath-sound are first distinctly recognizable. In this way an outer circle is drawn, marking off a larger area.

Further, the limits of dullness should be defined, and the position of other organs, the heart and diaphragm especially, taken into careful account.

Surgical Exploration.—The physician having thus marked out the area for exploration as accurately as possible, I believe it to be the best practice for the surgeon to make an incision along the lower margin of the intercostal space, which crosses the centre of the area marked. Having stopped all bleeding and fairly exposed the intercostal space, a puncture should be made with a fine silver canula through the central part of the inner circle, pushing the trochar boldly in, and quite vertically to the surface at the spot chosen. Several points may be thus explored should the first puncture not be successful.

The sign of success is the escape of some purulent secretion through the canula, showing that the cavity has been struck; the secretion may slowly well up, or be shot out with a rush of fœtid gas. The instrument should be introduced for an inch and a half or two inches in the first place, and the stilette then withdrawn for a few minutes; if no matter appear the instrument should be pushed in further, and then slowly and cautiously withdrawn, the surgeon testing each depth from the surface and endeavoring to ascertain by lateral movements the resistance of tissues and degree of fixity of the instrument. The length of the canula should be known beforehand, so that its depth of penetration may at any time be calculated. A small syringe or aspirator should be at hand, which may at any time be fitted to the canula.

Having failed to strike the cavity it is probably best to desist from further measures, but a fearless and thorough exploration should be made at several points before accepting failure. A silver canula is useful since it becomes readily discolored on meeting with gangrenous tissues or the fœtid contents of a cavity. The advantages of incising first the chest wall down to the pleura are great. The depth of the external tissues to be otherwise traversed by the trochar, unless this precaution be adopted, is considerable and uncertain, and the tightness with which the instrument is grasped by the skin renders it difficult or impossible to gain information with regard to resistance or otherwise within the chest. Only a rough guess can even be made as to the extent to which the lung has been penetrated.

Further steps in the operation.—If the escape of a few drops of discolored fluid or pus show that the cavity has been reached, the canula must be most jealously maintained *in situ* by an assistant, and used as a guide in further steps.[1]

The next point to decide is whether there is room for the drainage tube, and if there be any doubts on this point it will be best at once to remove an inch of rib before opening the pleura.

The costal pleura having next been divided for an inch, it will be at once seen whether the visceral pleura be adherent. In most cases it is so, and a narrow-bladed bistoury can be guided along the exploratory canula, so as to divide the inner pleural layer, and at the discretion of the surgeon to pass onwards until the cavity is reached. Only a slight incision need be made with this instrument, however, and it can then be withdrawn, to be replaced by the narrow probe-pointed forceps of Lister, passed along the directing canula until the cavity is reached, then opened out and withdrawn, so as to rend open the cavity with the least risk of serious hemorrhage.[2] The finger can now be inserted, the cavity thoroughly explored, its dimensions in different directions ascertained, and a large drainage tube introduced.

After the remarks already made in connection with empyema, it is scarcely necessary for me again to insist upon the importance of all instruments used, of whatever kind, being thoroughly clean and steeped in 1 in 20 solution of carbolic acid. The hands of the operator and of all those who have to do with the case must be carefully rinsed and all sponges wrung out of a similar solution of strength 1-40. The spray should also be used during the operation. If this should not be deemed necessary on account of fœtor being already present, it is well, as recommended by my colleague, Mr. Godlee, to thoroughly mop out the wound with a 1 in 40 solution of chloride of zinc, which has the additional advantage of arresting capillary hemorrhage. The usual charpie and carbolic gauze dressing is further requisite, allowing a sufficient thickness for the discharges to drain into.

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[1] In the discussion on Dr. Cayley's case of gangrenous cavity of the lung cured by external drainage, Mr. Godlee suggested the use of a canula devised by Sir J. Fayrer for exploring abscess of the liver, and provided with a groove for the purpose of acting as a director to guide any instrument to the cavity. Proceedings of the Roy. Med. Chir. Soc., N. S., vol. i., No. 6, 1884.

[2] Prof. Mosler of Greifswald, Ueber locale Behandlung von Lungenkavernen, Berl. Klin. Wochenschrift, Oct., 1873, was the first I believe to advocate the method of opening pulmonary cavities by the use of dressing forceps.

Smart hemorrhage will sometimes occur during this operation, as might be expected, but on air being freely admitted it usually soon ceases, if not, the wound must be for a short time plugged. I have not yet seen more than enough to cause momentary anxiety. Still it is most desirable to avoid as much as possible the use of cutting instruments in dealing with the lung.

In some cases, although the fine trochar gives evidence of having reached the cavity, it cannot at the time be found with the coarser instruments, the guiding canula having perhaps become displaced. Under these circumstances it is best to leave in a drainage tube, in the hope that the cavity may finally discharge through it. This happened in a most successful case of Dr. Cayley's, operated upon by Mr. Gould.[1]

The treatment subsequent to the operation must be carried on in the same carefully antiseptic manner, and in accordance with ordinary principles, and the special circumstances of each case.

In cases in which, on cutting through the parietal pleura, the lung is found not to be adherent, it is best to postpone the further steps of the operation for a few days, by which time adhesions will have become sufficiently established.

The danger of finding no adhesions is naturally much greater in recent than in chronic cases, and if the operation be persisted in, there is much risk of a virulent pleurisy being set up by the escape of some of the fœtid contents of the cavity into the pleura. It is best therefore, if possible, to wait a few days before proceeding further.

I have elsewhere[2] pointed out the importance of making the puncture over the point which the physical signs indicate to be the central part of the cavity, otherwise risk is run of missing it altogether, or of opening up some outlying loculus, which retracts and interrupts free drainage.

I have already intimated under their several headings, that neither suppurative nor gangrenous cavities require as a matter of course immediate interference; the abscess may discharge or the sphacelus escape through the bronchial tubes, and the patient make a good recovery. The physician must judge by the general principles already laid down, as to the necessity of interference. In diffuse gangrene, but little could be hoped for from surgical treatment.

The surgical treatment of bronchi-ectatic cavitation of the lung has hitherto, it must be confessed, been only partially successful, but where the area of disease is definable, and the patient's health is becoming undermined in spite of all the resources of medicinal and climatic treatment, operative measures may be advised, and I believe good may be effected, although the cavity or rather area of excavation be not thoroughly drained, by setting up contractile changes in the track of the drainage tube.

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[1] Med. Chir. Trans., vol. lxvii., also discussion in Proceedings, N. S., vol. i., No. 6.

[2] Med. Chir. Trans., vol. lxiii., p. 351, 1880.

In a case which occurred in my practice at the Middlesex Hospital in 1879, the first I believe of the kind in which the operation was performed, I was much struck by the extraordinary effect draining the cavity had upon the cough and expectoration. Although the amount of discharge though the drainage tube did not exceed two ounces, the expectoration, which had previously amounted to sixteen or twenty ounces per diem, was reduced to almost nothing.[1]

The surgical treatment of phthisical cavities offers less prospect of advantage than in other cases, and chiefly for these reasons. *Firstly*, the cavities are usually situated at the apex, and their drainage *per vias naturales* is therefore fairly maintained, so that the reason for interference is in the majority of cases not present.

Secondly, the cavities are rarely single when associated with any activity of symptoms, there being usually other smaller cavities in process of formation and coalescence at lower portions of the same or in the opposite lung.

Thirdly, experience teaches that completed excavation in phthisis is often the prelude to changes of a healing kind, resulting in more or less contraction of the cavity, drying of its secretion, and amendment of all symptoms. In fact, excavation in phthisis is a conservative process in so far as it results in the removal of morbid materials that are far more injurious than vacuity: half the history of phthisis is made up of this elimination process with which the hectic phenomena are identified, and with the accomplishment of which they cease if the perfected elimination be not overlapped by activity of process in other centres.

Hence pulmonary surgery with regard to phthisis has not I fear a hopeful future. Apex cavities have, however, been tapped and aspirated, and iodized solutions injected into them,[2] and although the results have hitherto not been encouraging, the operative measures have been generally well borne. Cases now and again present themselves in which the extent of excavation, superficialness of cavity, restriction of disease to one side, and large amount of secretion, strongly suggest external drainage, and I have seen indications after death of an attempt of such cavities to find an external vent by perforation of the chest wall.

An interesting case was recorded by Dr. Hastings as long ago as 1844, in which a large apex cavity on the left side was incised through the second intercostal space, and a drainage tube inserted, with immediate relief to cough and expectoration.[1]

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[1] On a case of basic cavity of the lung treated by paracentesis, by R. Douglas Powell, M.D., and R. W. Lyell, F.R.C.S., Med. Chir. Trans., 1880.

[2] F. Mosler, Ueber locale Behandlung von Lungenkavernen, Berl. Klin. Wochenschrift, October, 1873. Pepper, Philadelphia Medical Times, March, 1874.

Sir Spencer Wells, in a letter to the *Lancet*, shortly after the recent discussion on the surgical treatment of lung cavities at the Medico-Chirurgical Society, alluded to the case of an old man still living, in whom in 1843 a pulmonary cavity opened naturally by perforating the chest wall in the axillary region.[2]

The proposed operation of extirpating diseased portions of lung in phthisis and other affections, cannot yet be considered as fairly within the range of practical surgery.

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[1] This case was under the care of Dr. Hastings and Mr. Storks, and was commented upon by Dr. Hocken, Lond. Med. Gazette, December, 1844.

[2] See Lancet, 1884.

CHAPTER XV.

ASTHMA.

On more closely regarding the several varieties into which asthma is divisible for clinical convenience, it will be observed that they represent merely etiological sub-divisions of a disease which consists essentially of *a paroxysmal dyspnœa from disturbed innervation of the bronchi.*

To discuss again the numerous theories that have been held respecting the true nature of this disease would occupy too much space. These theories have been well related in Salter's[1] standard work.

The essentially spasmodic nature of asthma was recognized by Laennec, in consequence of the want of physical signs indicative of chest disease in the earlier periods of the malady, and from the fact that Andral had made some post-mortem examinations without finding anything in the lungs to account for the paroxysms. The character and seat of the spasm were more precisely ascertained by Dr. C. J. B. Williams[2] from his carefully devised experiments showing the contractility of the bronchial tubes under various kinds of excitation.

The anatomical and physiological facts upon which the bronchial spasm theory—now generally, but not universally, accepted—is based, are beyond dispute, viz.:—1. That the bronchial tubes are more or less muscular down to their terminations in the infundibula or alveolar passages (Schultze).[3] 2. That the bronchial tubes are capable of contractions, even to closure, in their smaller divisions, under electrical and mechanical stimuli (Williams). 3. That the nerve supply of the bronchial membrane is derived from the vagus and sympathetic, through the medium of the pulmonary plexuses and of ganglia distributed, to an undefined extent, along the course of the bronchi. (See page 15).

The clinical facts and arguments from which this view finds support may thus be summarized:—

1. The fact that an attack of asthma may supervene in the course of a few minutes, or even seconds, in a person in whom there can be found no evidence of chest disease.

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[1] On Asthma, its Pathology and Treatment, by Henry Hyde Salter, M.D., F.R.S., 1868.

[2] Experiments on the contractility and sensibility of the lungs and air tubes, 1840, see Diseases of the Chest, by C. J. B. Williams, M.D., 4th

[3] Stricker's Human and Comparative Histology, (New Syd
p. 60, 1872.

2. Of the organic lesions which can be said in different cases of asthma to have led up to the spasmodic seizures, there are none which singly or combined are proper to that disease.

3. Asthma leads on to certain pathological conditions recognizable during life, notably emphysema, chest deformity, cardiac disease, and visceral congestions; but these lesions are distinct mechanical consequences after long continuance of the disease, and in their earlier stages are totally inadequate to account for the asthmatic phenomena.

4. In view of the fact, which cannot be gainsaid, that asthma often occupies that period in the morbid history of cases, which intervenes between the occurrence of some inflammatory chest lesion and the supervention of marked emphysema and its associated phenomena, it has been maintained that asthma is a mere symptom, and its antecedents and sequelæ form one continuous though protracted pathological process.[1] The occasional occurrence, however, of asthma, without any pre-existing lesion, the frequent subsidence of the dyspnœic phenomena whilst the lesions remain, and the total inadequacy of any one or any combination of these lesions to give rise to the asthma paroxysm, compel us still to look beyond the pulmonary system for the pathology of the disease.

5. The family histories and mental proclivities of many asthma patients, the capriciousness and intermittent character of their attacks, recall to mind such affections as neuralgia, migraine, epilepsy. To how many causes may an attack of neuralgia be traced!—dental irritation, loaded rectum, fatigue, mental exhaustion, exposure to chill, disordered stomach, may be enumerated amongst them, yet the neuralgia is independent of them all, having its pathology apart; nay more, it may in turn produce certain peripheral affections, of which urticaria, herpetic eruptions, and local sweatings are examples. It will similarly be found that although many pathological states may be associated with asthma, the discovery of which may be of great value in directing treatment, yet the disease is a thing apart from them all, a disorder of the nervous system.

ETIOLOGY.—A considerable number of cases of asthma are attributable to inherited predisposition, and in many instances what is termed a neurotic family history obtains, although direct inheritance of asthma may be wanting. Epilepsy, insanity, neuralgia, chorea, hysteria, and asthma, being diseases all within the range of interchangeability in families.

Asthma is said to be much more common in males than in females. This does not happen to be in accord with my own experience. The disease is again stated by Dr. Salter and others to occur most frequently in the first decade of life, a period during which the maladies commonly calling it forth, prevails, *e.g.*, measles, bronchitis, whooping cough.[2] Between puberty and early middle life, and in females about the menopause, the disease is not uncommonly first manifested.

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[1] Berkart, On Asthma, 1878, p. 111. All that can be said in favor of the organic as against the nervous theory of asthma, is ably adduced and argued in this volume.

[2] Berkart, p. 127.

A depressed state of the general health predisposes to the occurrence, perhaps one might better say to the recurrence of asthma; but there are many individual exceptions to this rule.

The *exciting causes of asthma* are:—(*a*) *Direct.* (1) Irritants of all kinds, in the form of organic and inorganic dust or irritating vapors; (2) climatic influences of most varied kinds, those most distinctly potent being associated with dampness, excess of ozone, insufficient ventilation; (3) inflammatory chest affections, especially bronchitis, chronic pneumonia, pulmonary fibrosis; (4) pressure upon a main bronchus.

In this group it will be observed that the irritation which occasions the spasm, is applied to the mucous membrane in the form of dust particles, irritating atmospheric conditions, inflammatory excitation, direct pressure, or, as in the chronic fibrous affections of the lung, from mucus collecting in the bronchial tubes.

(*b*) *Reflex excitation.* (1) Gastro-intestinal disturbance, especially flatulent distension of stomach or bowels, and loaded colon; (2) intestinal parasites; (3) excitation at some higher point of the naso-bronchial tract;[1] (4) uterine disturbances, menstrual and climacteric; (5) excitation of cutaneous nerves. In these cases the broncho-pulmonary distribution of the pneumo-gastric and sympathetic nerves, is affected reflexly by irritation applied at some distant distribution of the same or of other nerves.

(*c*) *Centric excitation.* (1) Pressure of tumors upon the pulmonary plexus or disease affecting the pneumo-gastric centre; (2) emotional causes; (3) morbid conditions of blood circulating through the nervous centres ("humoral" asthma).

The production of asthma in association with morbid conditions of blood is probably of very complex mechanism. It is said that in those subject to asthma the circulation of blood tainted by the products of incomplete secondary digestion, causes the attack through direct irritation of the broncho-pulmonary nerves. Flatulent dyspepsia and irritating intestinal secretions are, however, inseparable concomitants of probably greater potency. Again, the asthma frequently observed in uræmic conditions is associated with pulmonary œdema, perhaps bronchial œdema also, and cardiac disturbance or disease.

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[1] Attention has recently been especially drawn to the fact that some cases of asthma are due to the presence of nasal polypi, and to a peculiar turgid condition of the turbinate bones, in fact to irritation of the sensory fibres of the fifth nerve, distributed to the nasal membrane. Hay asthma is probably in a great measure due to this reflex irritation. Dr. Semon refers to the observations of Dr. Hack of Freiburg with commendable caution in the Year-book of Treatment, 1884, p. 284. From Dr. Semon's observations it appears that the turbinates are y no means the bodies especially concerned in the production of the numerous lex maladies, asthma, cough, vertigo, epilepsy, etc., attributed to them by this ior.

(*d*) *Eruptive affections of the bronchial tract.* Asthma has been frequently observed to be associated with cutaneous affections, especially eczema and psoriasis. Sir Andrew Clark [1] has recently pointed out its frequent concurrence with urticaria, and contended that the phenomena of asthma were *mainly* due to urticarial-like swellings of the bronchial mucous membrane, such as occur in hay fever, with which the asthma often alternates, and out of which it often comes. I am convinced that a certain proportion of the cases of asthma I have met with may be thus explained. It is to be observed, however, that urticaria is itself a nervous lesion usually of reflex origin.

SYMPTOMATOLOGY.—The subjects of asthma present in the advanced periods of their malady a characteristic physiognomy:—thin, of nervous temperament, grave-featured, with slightly depressed angles of the mouth, high-shouldered and round-backed, they carry the impress of suffering in feature and build, and one is sometimes surprised at the power of work, keenness of wit and capacity for enjoyment with which these persons are gifted. It requires many severe attacks, however, to bring about these characteristic appearances, and in the intervals between the earlier seizures, there may be nothing in the physiognomy of the patient symptomatic of asthma. In the latter periods of the disease, and at an earlier date, after having recently passed through an attack, there may be quickened breathing accompanied by slight wheezing, very perceptible to the observer although the patient would consider himself quite free from dyspnœa.

There is another type of asthmatic, which may perhaps be distinguished as the gouty type, in which the patient is full-fleshed with excess of adipose tissue, especially about the abdomen, the general build being rather of the short-necked apoplectic type, than of the thin stork-like look of the victim of the neurotic form of the disease. The distinction is one of considerable practical importance.

Attack.—Patients with true spasmodic asthma may be seized at any time of the day or night, but more frequently the attack comes on at night after the first sleep. A certain feeling of oppression about the chest attended perhaps with some wheezing may give warning of the approaching seizure. More rarely the attack comes on almost instantaneously, and without warning. The sense of oppression may partially awaken the patient or give rise to some disturbing dream, and he either starts up in a fright with the fit of dyspnœa full upon him, or more gradually awakens to the increasing difficulty of respiration.

A severe attack of asthma is to the inexperienced truly alarming to witness; the expression of face, pale, staring, anxious and distressed, the mouth slightly opened, its corners twitched downwards with each brief effort at inspiration, whilst the neck muscles start forward in violent convulsive action. The shoulders are rounded, the body inclined forwards, and the hands rigidly grasp some firm object to fix the scapular and humeral attachments of the chest muscles. With the powerful inspiratory jerk thus effected the thorax is lifted *en masse*, but the deepening of the supra-clavicular hollows and depressed lower thorax, sternum, and epigastrium, bear witness to the small penetration of air to the lungs in response to all this effort. Expiration is still more difficult, for the expiratory force is, so to speak, beaten in detail. The air current in inspiration starts towards points of obstruction with a force proportional to expansion. In expiration, however, each infundibular current is obstructed whilst yet a feeble stream, the expiration is thus prolonged, labored, and but feebly and gradually effected, when, without a moment's pause, the quick, short, powerful, inspiratory jerk again takes place. The actual number of respirations per minute may not be increased, the pulse is, however, invariably quick, small, and often irregular and vacillating.

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[1] Paper read before the Bath Meeting of the British Medical Association, 1882.

In earlier attacks, more especially, there is great restlessness and frequent change of posture, with a disposition to lose self control in the desperation of air hunger. But the experienced asthmatic assumes and maintains some favorite attitude. The severity of attack may last from a few minutes to many hours, but it will be observed that even in the most urgent cases there are intervals of partial relaxation, during which the breathing becomes more easy, to be followed shortly by full intensity of spasm.

Towards the close of the attack cough comes on, and with the expectoration of some viscid mucous pellets, the dyspnœa is greatly mitigated. The body temperature during the seizure is depressed, cold sweats break out over the forehead, and the features become dusky, and partially cyanosed. In very severe cases capillary hemorrhages into the conjunctiva have been noticed (Walshe). The mind rarely becomes even for a moment clouded, however, and only in early cases does the patient sometimes lose that self-control which is of so much service to him in the struggle. The urine is generally abundant, pale, and of low specific gravity, and the solid ingredients are lessened (Ringer). The subsidence of the attack is generally gradual but sometimes almost sudden, with more or less expectoration, and the exhausted sufferer falls into a troubled sleep.

Physical signs.—During the attack the thorax is semi-expanded, with but little movement in response to the respiratory efforts, the percussion-note is resonant, and more or less of the emphysema type. Careful percussion is, however, impossible and useless. Auscultation reveals but little or no breath-sound, beyond a short wheezing in response to the sharp inspiratory jerk, whilst the expiratory murmur is wholly obscured by the prolonged cooing sibilus of varying note. The heart's impulse can be best felt, and sometimes strongly so, at or below the ensiform cartilage.

The amount of expectoration varies with the intensity and duration of the attack. The most characteristic expectoration consists of little pellets about the size of a pea, and "of the consistence of jelly or thick arrowroot, of a pale grey color, of an opalescent transparency, and a saltish taste" (Salter). These pellets are constituted of a viscid homogeneous matter, containing peculiar spheroidal or elongated cell-like bodies, of the size of large pus cells, but without nuclei, and with no distinct membrane. More or less numerous pigmented granular cells are also to be seen. Slight hæmoptysis is occasionally, but rarely, observed in severe cases of asthma. Where pulmonary œdema or congestion follows upon severe attacks of asthma, the expectoration is more abundant, consisting of a frothy sometimes slightly blood-stained mucus. Fibrinous casts of tubes may be found in asthma sputum, but are not constantly present.

After a severe attack of asthma, much prostration and fatigue is experienced from the severity and duration of the struggle, and from want of food and sleep. These symptoms will be soon recovered from, however, but renewed attacks commonly ensue at short intervals, until at the end of a certain series the malady appears to be for a time exhausted.

More or less wheezing rhonchi generally persist for some hours or days, and in severe cases it is usual to find at the bases some fine bubbling râles. The percussion signs and chest conformation at this period are those of acute pulmonary emphysema, varying in degree according to the severity of the attack. After a succession of attacks there is an amount of acute emphysema of the lungs with œdema of their bases, and overstrain of the right ventricle of the heart, requiring some time and treatment to remove. From single attacks, even although very severe, the patient after a night's rest may feel quite restored. In other cases again, the attacks are brief, but return each night; these are cases usually in which the paroxysm is controlled by some remedy, and although jaded by disturbance of rest, such patients are able to pursue their daily work or pleasures.

Periodicity of seizure is usually a marked feature of asthma. The seizures may be daily, weekly, monthly, yearly, or at other tolerably regular intervals. A patient may suffer a series of daily seizures, and then enjoy a certain interval of freedom. It will be observed, however, with asthma as with epilepsy, that as time advances the attacks, unless influenced otherwise by treatment, tend to become more frequent although less severe, a number of minor seizures being interpolated between the more regular attacks, the gradually increasing emphysema rendering the intervals less and less defined.

In the earlier years of true spasmodic asthma, patients are able in the intervals of attack to take part in sports and exercises, which make large demands upon the respiratory powers. As the disease continues from year to year, however, it gradually entails other symptoms significant of definite pulmonary lesions; shortness of breath, and more or less wheezing continue through the intervals: the physical signs of emphysema remain permanently, and visceral congestion and associated dyspeptic symptoms become manifested. The heart especially suffers, its right ventricle becomes dilated, systemic venous fullness ensues, and, finally, œdema of extermities, abdominal dropsy with enlarged and hardened liver, and albuminous urine supervene, (see Emphysema, p. 119). In a certain number of cases of asthma, on the other hand, the lungs give way, an atrophous form of emphysema appearing, which proceeds to coalescence of vesicles with adjacent pulmonary thickening, bronchi-ectasis and a spurious kind of phthisis at one or both apices. In yet other cases, in young subjects, the asthma is superseded by catarrhal phthisis, with the onset of which the paroxysms of asthma may wholly cease.

The *prognosis* of asthma proper is estimated by ascertaining the amount of physical damage the patient has as yet sustained to lung and heart. Asthma *per se* never kills, nor is the asthmatic prone to some of the diseases, *e.g.*, tubercle, cancer, Bright's disease, which shorten life.

His own peculiar malady is enough for him to contend with, and it enforces upon him a comparatively sheltered life. Asthma is in fact compatable with a life of medium length and of much usefulness, but of much suffering and self-denial. Family longevity should be inquired into in regard to prognosis.

For purposes of clinical classification asthma may be conveniently divided into:—

1. *Idiopathic or true spasmodic asthma.*
2. *Catarrhal asthma*:—
    Simple. Dust.
    Bronchitic. Hay.
3. *Peptic asthma*:—
    Gastro-intestinal.
    Humoral or metabolic.
4. *Cardiac asthma.*
5. *Eruptive asthma.*
6. *Uræmic asthma and other toxæmic forms.*

1. *Idiopathic or true spasmodic asthma* includes those cases of true neurosis in which no lesion can be found and in which the exciting cause of the attack, if any can be found, is either some mental or emotional disturbance, *e.g.*, mental shock, violent emotion, severe anxiety, or if material, is of so slight and ephemeral a kind as, *e.g.*, a brilliant light, a transient odor, the momentary application of cold to the surface, and the like. This form of asthma, as already said, can be best compared to neuralgia, epilepsy, migraine, perhaps also mania. The neurosis is inherited

either directly or through some of the allied forms mentioned. The attacks are more distinctly periodic than in any of the other forms of asthma, and with the completion of each attack the peculiar neurosis often seems to be for a time worn out.

2. *Catarrhal asthma.*—In this variety catarrhal symptoms precede and attend those characteristic of asthma, the dyspnœic phenomena being due to direct catarrhal irritation of the bronchial nerves, to reflex excitation of them through branches more highly distributed to the main bronchi or larynx, or lastly to similar excitation through the medium of the fifth nerve distributed to the nasal mucous membrane.

Bronchitic asthma is but a catarrhal affection of the bronchial tubes in a subject predisposed to asthma; the special character of the disease being derived from the individual peculiarity, not from the catarrhal affection, which commonly arises in the ordinary way.

In this affection during the acute stage the dyspnœa is more or less continuous. Subsequently, with the greater freedom of secretion and expectoration, the dyspnœa occurs chiefly in the early morning after a few hours' sleep, and is due to accumulation of mucus in the tubes, with the expulsion of which the spasm ceases.

In many of these cases there is some evidence to be found of a former inflammatory chest attack.

In cases of old standing emphysema fresh bronchial catarrh is often attended with dyspnœal seizures of an asthmatic type, which are due to obstruction of the tubes by mucus disturbing the, at all times, unstable respiratory equilibrium of the subject of emphysema. Only in popular parlance can such cases be called asthma.

Of *dust asthma* I have detailed a well-marked example arising from the inhalation of wood dust; other mechanical irritants have similar effects upon a certain proportion of those exposed to them (see page 107).

Hay asthma is a disease in which catarrh of a very intense kind is set up in the nasal and upper bronchial passages by the inspiration of pollen dust from graminaceous plants (anthoxanthum odoratum, species of Poa and other varieties). The perfumes of various flowers and the powder of ipecacuanha are said to affect sensitive persons in a similar manner.

Only a small proportion of those who suffer from "hay fever" or summer catarrh are afflicted with asthmatic paroxysms; and it is probable that the hay or other specific emanations produce asthma in a person predisposed to that malady after the manner of other irritants.

3. *Peptic asthma* is placed by Salter as a sub-variety of excito-motory or reflex asthma, in which the asthma is occasioned by "irritation of the gastric filaments of the pneumo-gastric nerve propagated reflexly to the pulmonary filaments, and producing through them motor phenomena, *i.e.*, bronchial contractions" (*loc. cit.*, p. 124). It is not to be doubted that

many cases of asthma are to be met with which are manifestations of dyspepsia, in the sense of some form of gastro-intestinal irritation.

This class of peptic asthma may, however, with great practical utility be enlarged to include cases in which faulty secondary digestion appears answerable for the incitement of the paroxysms. Dr. Salter expressed the belief that the introduction of food frequently gave rise to asthma, not by irritation of the alimentary canal, but by absorption into the veins of materials perfectly normal to the stage of the digestive process, but which in the asthmatic stimulated the unduly sensitive pulmonary nervous system to produce spasm (p. 32). Certain articles of diet, especially saccharine and carbonaceous matters, prove especially irritating to some asthmatics. In the incomplete metabolism that is a notable phenomenon in the gouty subject, the blood circulating through the lungs is also charged with imperfectly changed and effete materials. It may well be believed, too, that in cases of loaded colon, asthma is sometimes set up by the reabsorption of putrefactive matters. Whilst it is possible that in this "humoral" form of peptic asthma, the offending material affects the periphery of the pulmonary nervous system, it seems to me much more probable that the central portion of that system takes cognizance of such matters, and is unduly irritated by them.

4. *Cardiac asthma* is a consequence of heart failure. In heart diseases of various kinds, fatty and senile degenerations, valve disease with incomplete or damaged compensation, paroxysms of dyspnœa are met with. Most commonly the attacks occur during sleep or towards the morning, when the blood pressure is naturally low. Signs of passive congestion of the bases of the lungs, and more especially on one side, may generally be detected, and some blood-staining of sputa is not infrequently observed to follow the attack.

5. In *uræmic* asthma, œdema of the lungs is generally present. I have seen cases, however, in which the morbid material in the blood seemed to be more directly the excitant.

6. *Eruptive asthma.*—Under this heading may be included those cases in which asthma is associated with cutaneous eruptions (especially urticaria) in which there is reason to suspect that the bronchial mucous membrane is involved.

TREATMENT.—There is no disease which is so extensively "quacked" as asthma. Persons who are the victims of the pure neurosis are amongst the most restless of mankind. Their temperaments are often of the highly-strung nervous type, and whilst they are gifted with much courage, endurance and determination, they possess little faith, many friends, and much credulity. The practitioner who would guide these sufferers, must himself have clear opinions respecting the salient points in treatment, and must be patient in hearing and endeavoring to understand the individual experience of the patient himself.

The treatment of asthma must be divided into climatic, anti-spasmodic and remedial.

Climatic treatment consists, firstly, in the removal of the patient from those external surroundings which appear to have led up to his attack, to more favorable conditions of residence, *e.g.*, from a dusty to a pure atmosphere, from a cold, damp house or locality to a dry soil and a well-ventilated and cellared house with no trees, unless they be of the pine tribe, in near proximity.

Secondly, to tell with precision what locality will suit a given individual with asthma is very difficult; there are idiosyncracies in each case, and no asthmatic should burden himself with a house until he has first tested the locality by residence there for some time.

There are, however, certain climates which are most likely to prove beneficial for asthma patients. (1) Bournemouth, the St. George's Hill neighborhood of Weybridge, Farnborough, and Bagshot districts, may be named amongst English localities, and Arcachon for the winter and spring abroad, as places which are under all circumstances preferable to cold, damp neighborhoods, but as being best adapted for the purest forms of asthma in which the neurosis is most marked.

(2) Torquay, St. Mary Church, Pau, Cimiez, Hyères, Algiers, are varied resorts adapted for winter residence for the mixed catarrhal forms of asthma.

(3) Experience teaches a certain number of asthmatics that a sea voyage does most for them, and in such cases the cure may best be thus started. Cases of dust and hay asthma are especially suited for this plan, as also are cases in which the first seizure has been traceable to a nervous system broken down by anxiety, overwork or excesses.

(4) In young subjects with as yet no marked emphysema, the rarefied airs of St. Moritz, Davos, Wiesen, yield good results, and in the hot months they may be resorted to with advantage by some older subjects. Ilkley, Ben Rhydding, and Dartmoor, are very suitable situations for convalescent asthmatics in the summer season.

(5) A considerable number of asthma patients do best in towns, being chiefly cases of those who have removed from more or less damp localities surrounded by trees. As a rule asthmatics (and indeed all chest invalids) should repair to large towns or sea-side resorts in the late autumn, and perhaps the late spring.

Medicated airs and baths.—In cases of catarrhal asthma and in those in which a gouty element, or an association with eczematous eruptions or urticaria, can be observed, a short, two or three weeks' summer course at Mont Dore, Aix les Bains, Allevard les Bains, or Dax, will be attended with long-continued benefit, the course at one of these bathing resorts being followed up by a further sojourn at some bracing place, *e.g.*, Eaux Bonnes, Cauteret, Spa, or the Swiss mountains, or returning home to

Ilkley, Ben Rhydding, Braemar, or other moorland districts of Yorkshire or Scotland. A course of Turkish baths in London, Ramsgate, or Ben Rhydding, for those who cannot get abroad, sometimes proves of great service. I have in some cases seen much benefit from the use of the compressed air baths at Ben Rhydding, but I can only mention this as an empirical fact. Minute traces of arsenic mingled with the vapors at the Mont Dore baths, are regarded as answerable for their good effects, and at Allevard the sprayed air of the inhalation chambers is decidedly charged with sulphuretted hydrogen. This latter treatment is most adapted for the chronic bronchitic forms of the disease; at the other places probably the sweating of the skin and bronchial membranes constitutes the chief remedial factor.

Regulation of the digestive function.—Not less important than the selection of a suitable climate, is the careful regulation of the digestive functions of the asthmatic. In a large proportion of cases the exciting cause of the attack is some error in digestion, whilst in all cases the digestive function ultimately suffers, and reacts unfavorably upon the spasmodic troubles. In cases in which there is excess of uric acid or urates, and a disposition to flatulent dyspepsia, cutaneous eruptions, etc., sweet wines, raw fruits, sugar, cooked butters, pastry, should be excluded from the dietary. Very slow eating should be strictly enjoined, and the staple food, including a moderate supply of meat, should be taken by the mid-day meal, only the lightest possible diet being allowed later in the day. An alkaline bitter may often be taken twice a day with great advantage when the tongue is red and coated, bismuth being added when there is gastro-dynia.

The liver and bowels require careful attention, very small doses of mercurial being useful from time to time, whilst in these, as in all neurotic subjects, large doses of this drug are positively harmful. A sulphate of soda saline should be taken in hot fluid in the morning after each dose of mercurial. If the rectum be loaded, relief should be effected by enemata rather than by violent aperients.

When the tongue is fairly clean, or at all events when the practitioner is satisfied that the *primæ viæ* are efficiently acting, a course of arsenic is often of the greatest service.

Treatment of the paroxysm.—Most commonly the practitioner makes his first acquaintance with the asthmatic patient when the paroxysm is at its height. A few questions as to duration of attack, preceding attacks, and circumstances and symptoms immediately antecedent to present seizure, will suffice to direct a rational and safe treatment.

When irritating matter is present in the stomach, whether in the shape of undigested food or irritant catarrhal mucus, an emetic of twenty grains of ipecacuanha, or a subcutaneous injection of $\frac{1}{10}$ to $\frac{1}{6}$ grain of apomorphia, will give prompt relief by its removal. Warm water, with a little carbonate of soda, should be given to encourage a thorough clearance of the stomach. In cases of catarrhal asthma during the paroxysm ten minims of ipecacuanha wine, with fifteen or twenty minims of ethereal tincture of lobelia, given every half hour for two or three doses, then every hour or two hours, will often prove serviceable. The fluid extract of Grindelia Robusta (U. S. P.) in fifteen minim doses, may be sometimes substituted for the ether.

In the choice of sedative remedies, a careful judgment must first be arrived at as to the amount of tubal catarrh and secretion present. The well-known "cures" of asthma, appear to owe their efficacy partly to an evacuant, partly to a sedative action. The fumes of the "Himrod," "Green Mountain," "Chester," and similar powders first excite more or less cough, after which, and especially if expectoration be effected, the spasm yields. A powder containing four drachms of powdered stramonium, two drachms of each of powdered nitre and aniseed, and five grains of tobacco is a very efficacious combination, much used at the Brompton Hospital. A teaspoonful of this powder should be made into a conical heap on a plate, lighted at the summit, and the fumes inhaled through a large inverted funnel. In the more purely nervous forms of asthma, when as yet there is no secretion present, more purely sedative remedies must be used.

By closely shutting the room and filling it with the fumes of nitre,[1] the asthmatic sufferer will sometimes gain relief and rest. Salter considered nitre thus used a powerful sedative; but it must be confessed that its mode of action is obscure; its efficacy in certain cases is, however, unquestionable. A little chlorate of potash may sometimes be combined usefully with the nitre (about one drachm to the ounce). In the full height and intensity of attack, iodide of ethyl, nitrite of amyl[2] or chloroform may be inhaled with temporary advantage, and the true spasmodic nature of the affection will be well demonstrated by the complete subsidence of the sibilus under the influence of chloroform. The effect of these drugs is as a rule very evanescent, however, and their use is only desirable to mitigate extreme symptoms. Nitro-glycerine and nitrite of sodium have been recommended.

Joy's cigarettes and Savory and Moore's datura tatula cigarettes will, provided the patient can inhale the smoke into the lungs, in many cases give great relief to the urgent symptoms, their efficacy being due to stramonium and, probably, some opium combined.

In severe cases an almost unfailing remedy is morphia used subcutaneously in doses of from $\frac{1}{8}$ to $\frac{1}{2}$ a grain. Morphia is a remedy which, from the immediate relief it affords, is apt to be given somewhat recklessly, and to be called for by the patient peremptorily without counting the cost in after consequences. The drug, however, should be used with caution, for not a few casualties have arisen from its employment. In catarrhal cases it should not be employed, and in the most purely neurotic cases, for the relief of which it is best adapted, it is a demoralizing remedy. In fact the employment of morphia in asthma is exactly comparable to its use in neuralgia; prompt in relieving, it lowers nerve tone, hampers secretion and increases liability to recurrence. The tendency of the patient is increasingly to fly to the use of the drug for trivial attacks, until custom renders a large and dangerous dose necessary. In certain cases the drug is necessary; they are exceptional, however, and in such cases the most strenuous efforts should be made by the practitioner to remove his patient out of the conditions to which the asthma appears attributable. Hypodermic injections of atropine $\frac{1}{100}$-$\frac{1}{80}$ gr. are sometimes useful, and are not open to the same objections as morphia.

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[1] Four ounces of nitrate of potash dissolved in half a pint of boiling water, pour out into a soup plate, and draw through thick blotting paper, dry and cut in squares of four inches.

[2] Convenient five-drop capsules of these last two drugs enclosed in silk are made by Martindale of New Cavendish Street, W.

Chloral in full doses of fifteen or twenty grains every four or six hours is also efficacious in pure asthma, and it may be used also in the catarrhal forms; its administration, however, requires strict medical supervision and very precise directions.[1]

In hay asthma Salter recommends tobacco smoking continued to the development of symptoms of collapse. The remedy is only serviceable to those unaccustomed to smoking.

In cases of catarrhal asthma especially, and in some other less-defined cases, one of the most valuable remedies is iodide of potassium, in combination with stramonium. The iodide is especially indicated in those cases in which there is a nightly paroxysm, but in which there is a perceptible dyspnœa and wheezing throughout the day.

Three to five grains of iodide of potassium, with $\frac{1}{4}$ grain of extract of stramonium, should be given every three or four hours during the day, *e.g.*, 8 a.m., 12 noon, 3 p.m., 6 p.m., 9 p.m., so as to administer some twelve to thirty grains of the salt, and two to three of the extract, by bedtime. After a day or two, when the patient suffers slight iodism and the physiological effect of the stramonium upon pupil and throat becomes apparent, the remedy may be continued in half doses. In the many cases in which this remedy proves valuable, it should be commenced on the first approach of asthma phenomena. In some cases in which asthma has been contracted in malarious climates, or in which a neuralgic element is traceable, quinine may be usefully given in combination with iodide of potassium. Five grains of salicylate of quinine in pill (with citric acid) may be taken three times a day, either at separate times or with each dose of iodide.

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[1] No doubt "Bromidia," consisting of chloral, gr. xv.; pot. bromid. gr. xv.; ext. cannabis ind., gr. $\frac{1}{8}$; ext. hyoscyami, gr. $\frac{1}{8}$, in each fl. ʒ j., will become much used in asthma.

Alcoholic stimulants should only be given in asthma when necessary as a restorative and in small doses. Strong coffee is of great value as a restorative after a severe paroxysm, and in some cases it distinctly relieves spasm; or caffeine, of which two or three pills of 2½ grains each, made with glycerine of tragacanth, may be taken for the same purposes. After an attack of asthma, especially if it has been severe and has left any pulmonary œdema behind, it is good practice to give small doses of digitalis (five to ten minims, three times a day) to restore tone to the heart and small vessels.

The digestive system requires careful looking to; the secretions will necessarily have become disordered by the drugs used for the relief of the paroxysms, and by the venous conjection of viscera necessarily attendant upon the impeded pulmonary circulation. It is probable, too, that the function of the pneumo-gastric nerve becomes seriously enfeebled for a time by the attack and the remedies. A little pepsine and hydro-chloric acid after food, in combination with nux vomica or quinine given an hour or two before food, prove very serviceable. The most careful consideration must immediately be given, however, as to whether the patient cannot be moved to a more suitable locality, and this may in some cases be wisely effected, even in the midst of an attack.

CHAPTER XVI.

ON THE PATHOLOGY OF PHTHISIS.

On inspecting the lungs of one who has died of phthisis, we meet with a very great variety of appearances, which may, nevertheless, be recognized as the results of a comparatively few morbid processes; we see consolidation of the lung in every stage of formation, decay, and removal; and, glancing at the emaciated form before us, we have a very practical definition of Phthisis Pulmonalis, viz.:—progressive consolidation and decay of the lung with progressive wasting of the body.

The exact nature of the morbid processes which lead to this destruction of lung and waste of body, whatever their etiological origin may be, are of two kinds. 1. Inflammation affecting with different degrees of intensity the different tissues of the lung, and running an acute, chronic, or chequered course. 2. A new growth—Tubercle—with its characteristic granulations disseminated through the lungs, or collected into nodular groups, or mingled with inflammatory changes, developing into fibroid tissue, or immediately undergoing necrotic change. We may meet with either of these processes in the acute or chronic form without any admixture of the other, but it is comparatively rare to meet with chronic tubercle unmixed with inflammatory changes.

It may be here recollected that fatty degeneration is one of the consequences of inflammation, and is the means by which the products of inflammation become eliminated or absorbed, or for a time sequestered by caseation.* The tendency of all new growth is also sooner or later to fatty decay, and in tubercle we find a special aptitude in this direction, it being a cellular neoplasm devoid of vessels. There are furthermore two conditions so inseparable from phthisis as to constitute a part of its pathology, viz.:—3. The respiratory movements of the chest cavity with their attendant mechanical effects upon the lungs and pleura under influences of disease. 4. The penetration of the lungs by specific and putrefactive organisms.

1. The inflammatory process takes the largest and most important share in the production of the various appearances met with in phthisical

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[1] It has been recently held that there is something specific in caseous change; indeed, that this change is at least in great part brought about by the action of minute organisms, or their germs, the bacilli of tubercle. I have failed in the endeavor to find anything tangible in this hypothesis.

lungs; it is the destroying element in this disease, as was long ago pointed out by Addison.[1] It may, therefore, be appropriately spoken of first.

With that form of inflammation of the lung—acute sthenic pneumonia—agreeing in many of its characters with an acute specific disease (*e.g.*, idiopathic erysipelas), we have but little to do in dealing with cases of phthisis; we only meet with it as an exceptional complication. A person affected with acute basic pneumonia who is already cachectic, or rendered so by neglect during the disease, may become phthisical; some cases of basic phthisis have this origin. But the pneumonia which is the most constant element of true phthisis, and which will indeed, as already hinted, be found the main factor in the more chronic and cachectic forms of basic lung disease, is of a very different kind; its onset is usually insidious, and its origin appears to be generally by extension of a catarrhal process from the finer bronchial tubes to the interior of the alveoli; hence its name—*catarrhal* pneumonia, or as it might be termed, bronchoalveolitis. This form of pneumonia is essentially lobular, although the coalescence of many adjacent lobules may cause the consolidation of a whole lobe. The alveoli are affected by this inflammation with all degrees of intensity, from mere superficial catarrh causing slight epithelial desquamation to the most deeply destructive involvement of their walls.

In the simplest alveolar catarrh the cellular products may escape with the expectoration, leaving the alveolar wall undamaged. In the next degree of intensity, the alveoli and minute bronchi become blocked with the large granular cells, which are produced in great abundance, and which may perchance be mingled with a few leucocytes. These cells, thus stuffing the alveoli, almost immediately begin to undergo fatty degeneration—the process by which resolution is naturally affected. They may liquefy, and be partially absorbed, partially expectorated; but the alveolar walls have been damaged, and permanent local collapse remains behind from their agglutination. This is the natural cure of the disease in this degree, the slightest degree that can be called phthisis.

More commonly the cellular products, after having undergone complete fatty degeneration, become inspissated by absorption of fluid matter, and remain for a long time—perhaps for the lifetime of the patient—in the cheesy condition, or subsequently become cretaceous. This may be called natural arrest by obsolescence, and these cheesy masses are commonly looked upon as "old tubercle."

In the still more intense degree of the process—catarrhal alveolitis—now under consideration, the alveolar wall is deeply involved in the inflammation, so that it subsequently undergoes fatty degeneration together with its cellular contents to an extent which varies with the intensity of the process, and breaks down in the subsequent liouefaction, gradually or rapidly according to circumstances.

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[1] Collected writings published by New Sydenham Society, p. 56.

The elastic tissue of the lung takes no active part in any of its inflammatory processes; it escapes but little altered when the alveoli break down, and thus, on being recognized in the sputa, affords certain evidence of pulmonary destruction. It will, of course, be understood that there is no real line of demarcation between the degrees of severity above described separately. The intensity of the first attack may at once determine the depth of injury, or the lighter may gradually pass into the graver degree.

But in addition to the parenchyma proper of the lung, which, with its epithelium, is the special seat of catarrhal pneumonia, there is the fibrous stroma, if one may so style it, formed by the interlobular areolar tissue, supplying sheaths to the vessels and bronchi, contributing also to the formation of the alveoli, and intimately connected at the surface of the lung with the investing pleura. It could not be expected that an inflammation of the lung of any great severity would leave this widely spread tissue untouched; and it might also be anticipated, on reflection, that a tissue thus, comparatively speaking, deeply placed would, as a very general rule, only be affected secondarily to disease of the parenchyma or pleura. From this interstitial tissue under conditions of inflammatory irritation are derived, in great part, the tough, fibrous, pus-secreting walls of cavities, and the trabeculæ which for a long time resist the most severely destructive processes. The inflammatory process in this tissue is, as a rule, a much more deliberate one; even when in a state of active ulceration, as in the walls of some cavities, the destruction is molecular, sphacelus is rare. Inflammatory reaction in this tissue, indeed, more generally partakes of the character of growth under irritation, producing a more or less general condition of *fibrosis* of the lung. Decay, however, in most instances, finally sets in; the fibrous tissue, at first merely hyperplastic, loses its characters as such; its nuclei, at first very abundant, gradually fade; its fibres fuse into tough homogeneous bands, and in their turn become granular and fatty, and finally crumble away.

The various primary diseases—catarrhal pneumonia, croupous pneumonia, chronic tubercle, pleurisy, etc.,—upon which pulmonary fibrosis supervenes, are thereby marked by clinical features of great interest and of significance for prognosis. But sometimes the fibrosis is so extensive as to become, whatever its origin may have been, the essential disease. Such cases have been very conveniently classified separately by Sir Andrew Clark under the term "fibroid phthisis." He regards the disease as sometimes of idiopathic origin, or—what amounts to nearly the same thing—as a disease which progressively invades and destroys the lung from some one point of origin, as a local pleurisy or bronchitis.

2. In order to apprehend the nature of tubercle, its relationship to other products of pulmonary phthisis, and some of the most important ways in which it extends and is disseminated, we must recall to mind that

the whole lung is pervaded in every crevice of its structure by lymphatic tissue, consisting of branched protoplasmic cells communicating with fine tubes and interstitial spaces occupied or lined with endothelial cells.[1] The characteristic form of tubercle is the grey granulation, a hard, semitransparent, cartilaginous-looking nodule of about the size of a pin-head, intimately connected with the surrounding tissue. Each granulation consists essentially of a collection of small, rounded, lymphoid cells with a delicate reticulated stroma, the cells being derived by multiplication principally from the lymphatic elements of the lung; no vessels are present in tubercle, although a vascular zone sometimes surrounds young granulations.[2] From their close contiguity to vessels, however, the nutritive supply of the granulations is assured until from the dense multiplication of cells their central portions suffer deprivation. In the centre of the small collection is usually to be found one or more "giant cells," irregular masses of protoplasm enclosing many nuclei, with a more or less branched outline, which blends with the stroma of the tubercle. Hamilton[3] regards the giant cell as an essential part of tubercle, and maintains that the peculiar fibro-cellular texture of the granulation is evolved by development of the periphery at the expense of the central protoplasm of the "cell." Ziegler[4] and others[5] are of opinion that there is nothing in the shape, size, or characters of the cells, absolutely significant of tubercle, the giant cells being found in many other morbid products (a fact which Hamilton admits), and not being necessarily present in the tubercular granulation. The grouping of the cells to form a definite nodule of a certain size which is avascular, and tends to necrotise in the centre, constitute the features peculiar to this neoplasm. Moreover, when subjected to proper staining processes, one or more tubercle-bacilli will probably be detected in the nodule, particularly within the giant cells. Whilst Dr. Burdon Sanderson has especially observed the sheaths of the minute bronchi as favorite sites for the origin of the tubercular granulations (there being, in the guinea pig at all events, minute gland acini in these situations), Dr. Wilson Fox has, on the other hand, pointed out a remarkable obstruction to the minute vessels of the lung as most characteristic of the tuberculizing process. Dr. Klein has also shown that the ultimate branches of the pulmonary artery become both invaded by the growing adenoid tissue from without, and thickened and obstructed at points by proliferation of the endothelial lining of their own inner coat. The alveolar walls, the peri-bronchial, peri-vascular and sub-pleural tissues, are then the seats of tubercular growths; but under special circumstances of irritation tubercle may be developed upon the surface of the pleura from the endothelial cells of the stomata; this growth may similarly intrude into the alveolar spaces from one point of the alveolar wall, as described and figured by Hamilton, p. 181, fig. 57, or possibly by outgrowth from the pseudo-stomatous processes which normally penetrate to the alveolar surface. It would seem then, that, broadly speaking, *the seat of tubercle is the interstitial connective tissue of the lung.*

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[1] For the development and further organization of this lymphatic, new growth is required "no more than the presence, at the seat of the irritation, of a pre-existing element of the group of connective-tissue corpuscles and endothelium." Buhl, quoted by Klein, op. cit., part ii., p. 69.

[2] Vide plate v., fig. xxiii. of Klein's work.

[3] Hamilton, Pathology of Bronchitis, Catarrhal Pneumonia and Tubercle, pp. 172-177, 1883.

[4] Ziegler, Pathological Anatomy, part i., p. 166, 1883.

[5] Prof. Sée, Phtisie Bacillaire, p. 42, 1884.

Under favorable circumstances[1] tubercle undergoes development into a peculiar form of fibroid tissue, at first very recognizable from ordinary hyperplastic fibrous tissue, but which subsequently becomes converted into bands or tracts of uniform homogeneous texture, and finally degenerates and is removed. This development of tubercle, before its final decay, has hardly been sufficiently insisted upon as an essential character always observable if circumstances permit the attainment of the necessary stage. It is, however, in strict accordance with the lymphatic gland type of this morbid growth, and it is of some importance as affecting the clinical characters of chronic tuberculosis. In acute tuberculosis, the patient does not often live long enough for any process of the kind to take place. In chronic pulmonary tuberculization, however, and when tubercle attacks a lung rendered quiescent by previous disease, the stages of development of tubercle into fibroid tissue may be seen.

The anatomical relationship of tubercle to phthisis and to tuberculosis respectively is a question upon which very conflicting opinions have been expressed. Until the time of Addison, and indeed up to the researches of Virchow upon tubercle, Laennec's views held good, and the tendency is now to return to them and to regard tubercle as the anatomical lesion of phthisis, and its caseation and softening as the cause of the lung destruction in that disease.[2] It was not long after Bayle's and Laennec's teachings became known before the "tubercle corpuscle" was discovered, and the revival of the views of the great French pathologist received support from the discovery of the "giant cell," still regarded by some as the criterion of tubercle. But unquestionably the pathological observations of Buhl, and the experimental inquiries of Villemin and his many followers, and finally the discovery by Koch of the tubercle bacillus have far outweighed all histological considerations towards resuscitating the doctrine of Laennec with regard to the essential unity of the lesions of phthisis. The difference histologically between tubercle and catarrhal pneumonia is, however, enormous, and it is to me inconceivable how the two can be jumbled together as one product, even though they are often found in close association under apparently similar etiological conditions.

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[1] The death and removal of tubercle may be greatly hastened by associated inflammatory processes.

[2] "L'unité de la phtisie comprenant toutes ces manifestations aiguës et chroniques; il n'existe plus de dualisme entre le tubercule et la pneumonie caséeuse, ni de distinction entre la phtisie turberculeuse et la phtisie inflammatoire, la pneumonie caséeuse ayant elle-même le bacille comme témoin constant, comme origine certaine ; la pathologie expérimentale en fournira la preuve."

If we regard the lymphatic elements which everywhere pervade the lung as the histological source of tubercle, it is easy to conceive that we may have tubercle not only in the form of isolated nodules (miliary tubercle), but also in a more diffused form thickening alveoli and consolidating the lung by more or less extensive tracts of adenoid tissue (diffused tubercle).

It is impossible on anatomical grounds logically to refuse this admission, and it is in this more liberal acceptation of the term tubercle by Dr. Wilson Fox, that one step on the return to Laennec's view may be regarded as having been taken. Let us, however, in this connection carefully note the difference between the terms *tubercle* and *tuberculosis*. Tuberculosis is a disease-process of the definite nature of which we are daily becoming better informed, tubercle is a disease-product. The process tuberculosis may be said to consist in the more or less wide or universal dissemination through the body of a poison-influence which results in an outbreak through the infected region of innumerable tubercle growths. But tubercle may arise locally by direct infective irritation, and may thence occur not in isolated granulations but in the more diffused form.

Hence there need be no ambiguity about the term tuberculosis, which signifies an acute and more or less general outbreak of miliary tubercle, whilst tubercle itself, which in the form of the miliary granulations is the essential lesion of tuberculosis, may yet arise locally, outside, as it were, the general system.

3. The respiratory movements of the chest cavity take part in the pathology of phthisis as of other chest diseases, but in a more marked degree.

The real explanation of the recurring pleuritic pains and adhesions in cases of phthisis is, that when a portion of lung becomes damaged in texture by disease it ceases to follow accurately the expansile movements of the chest-wall; a certain gliding or rubbing motion takes place between the two normally corresponding pleural layers at this point;[1] friction, local pleuritis, and adhesion result. We can readily understand, therefore, how it is that a friction sound is often the first evidence we get of local pulmonary disease, and that a new friction sound generally means more than mere dry pleurisy; it means, in fact, an accession of lung disease.

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[1] Most authors state that a gliding movement naturally takes place between the two pleural surfaces. I am myself inclined to think that in perfect health no such motion occurs, but that the expanding lung accurately follows the expansion of the chest. Whether this correspondence in expansion be perfectly accurate or no, the gliding and friction must be greatly exaggerated over the region of a diseased portion of lung, and the explanation of adhesions in the text remains good.

When the lung disease is of a very chronic, indurative, contractile character, the effect of the continued inspiratory efforts to expand the toughened lung is to stretch out the adhesions and to separate the pleural layers to a certain extent; the further contraction of the lung continues the process, so that the parietal and visceral pleuræ may become separated by a considerable interval of half or three-quarters of an inch. This space is at first filled by serous fluid effused into the meshes of the areolar tissue of the stretched adhesions. We thus get the œdematous pleura. At a subsequent stage, however, of the disease, by the continued growth of the areolar tissue, the whole space becomes occupied by tough fibrous tissue, and the two layers become completely welded together into one uniform fibrous thickness. That this is the real history of the enormous thickening of the pleura in many cases of chronic phthisis I have satisfied myself by repeated observation.[1]

It has seemed to me that thickening of the pleura has been regarded too much in the light of a dangerous pathological process, liable to extend into, and by its contractile power to compress, the proper lung tissue, whereas it will be found on careful examination to be most generally a condition secondary and quite subsidiary to the lung disease.

In primary pleuritis the thickened pleura is produced in a different way. After absorption of the fluid a certain thickness of lymph often remains between the two layers of the pleura, into which the granulations from each surface penetrate, and finally unite, completing the adhesion. There are many cases of phthisis of the pneumonic kind of tolerably acute progress, and attended with little contraction, in which, though the pleural surfaces are inflamed and covered with finely granular lymph, they do not become united. It is in these cases that pneumo-thorax is especially likely to occur.

The constant movement of the lungs no doubt goes far to modify and hasten the progress of morbid processes going on within them. Let us think of the lung roughly as an intricately infolded membranous surface, closely allied to, and continuous with, the bronchial mucous membrane, but peculiarly rich in blood vessels and lymphatics. The products of a catarrh affecting the larger bronchial tubes can be readily expelled without difficulty or danger, but a much greater difficulty attends the removal of the products of a similar affection of the alveoli. Indeed, it is accomplished only in small part by expectoration; those portions which are not readily liquefied and absorbed are apt to accumulate, and by their subsequent decay to irritate the alveolar wall and to set up in it those proliferative and inflammatory changes which constitute local "tubercle"—just as the retained secretions of a sebaceous follicle may give rise to the acne pustule. The thickened alveolar walls in their turn degenerate and soften or suppurate, and in these few changes occurring in numerous centres corresponding to the lobular groups of alveoli, we find the very basis and foundation of the chief morbid results of phthisis. Add to this group of changes the definite growth, miliary tubercle, which may arise in the neighborhood infected from the caseous foci, or which may spread throughout the system from such centres;—in a word, add a description of what we understand by the term *tuberculosis* to the phthisical changes we have enumerated, and all the active processes which produce pulmonary destruction in nine out of ten cases of consumption are summed up.

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[1] Vide Trans. Path. Soc., vol. xx., pp. 59-61.

The ceaseless movements of the lungs and the constant access of air to the diseased surfaces, keep up the suppuration and maintain the tendency to the absorption of septic matters, and by the inhalation of portions of sputa, laden with acrid and specific poison to distant parts of the lungs, the lesions of phthisis are further propagated and disseminated.

4. The penetration of the lung by specific and putrefactive organisms thus brought about results in changes of two kinds, each very decidedly influencing the clinical features of the disease.

(*a*) In the excavation stages of phthisis the morbid secretions are necessarily invaded by putrefactive bacteria.

(*b*) A special organism regarded as peculiar to phthisis, finds its way chiefly by inhalation, sometimes also by other routes to the lung. It is held by some observers to be primary to the lesions of phthisis, to be indeed the germ of that disease, others again regard it as an epiphyte to the generation of which the phthisical soil is specially favorable.

CHAPTER XVII.

ETIOLOGY OF PHTHISIS.

THE tendency of modern inquiry is to range all the conditions which have been hitherto regarded as causative of phthisis on the side of *predisposition, i.e.*, as conditions which bring about a state of receptivity, if I may say so—a capacity on the part of the tissues to receive and harbor with hospitality a certain organism, and on the other hand to regard the *exciting cause* as one and single—that organism, the tubercle bacillus.

That there are difficulties in the way of accepting this view I shall point out, but it is based upon the results of careful observation and researches extending over many years.

It is impossible at the present time to discuss the etiology of phthisis without first facing this question which is at the root of the matter, viz.: —The relationship of the bacillus tuberculosis to the disease.

The specific nature of tubercle which was believed in by Laennec and by others long before his time, may be said to have been first demonstrated pathologically by Buhl in 1857, when he pointed out that an outbreak of tuberculosis was almost always attributable to the previous existence of caseous matter somewhere in the body. Villemin advanced the question a step further in 1865, by showing that caseous matter introduced into a healthy animal, produced tuberculosis, and that therefore the tubercular virus dwelt in caseated products. The announcement of Villemin's discovery by Mr. Simon from the chair of the Pathological Society of London, was followed by numerous experiments, confirmatory and otherwise, by competent observers in this country; the general result being that Villemin's conclusions were substantiated. Some further experiments suggested that matters other than tubercular, when introduced into animals predisposed to tubercle, set up at first local, and then more diffused tubercle, but quite recent researches point to the probability of there having been some fallacy in these experiments; for due precautions being taken to use perfectly clean instruments and vessels, and to exclude all tubercular animals from the neighborhood of those under operation, inoculation with materials other than tubercular failed to produce tuberculosis.[1] Fresh point and precision were given to the conclusions of Villemin by Koch's discovery in 1882, of the presumed morbific agent in the transmission of tubercle, viz., the *bacillus tuberculosis.* In this bacillus, in the opinion of Koch and his followers, dwell the virus and potentialities of tubercle.

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[1] For original experiments bearing upon this point, and for an admirable resumé of the recent literature of the subject, see paper by Dawson Williams, M.D., in the Pathological Transactions, vol. xxxv., p. 413.

The main facts with regard to the life history and potentialities of the tubercle-bacillus may be stated as follows:—

(1) The tubercle-bacillus is a minute rod-shaped fungus, measuring from 0·003 to 0·0035 millimeter in length, and about one-third that measurement in thickness. The rods are straight or slightly curved with rounded ends, and often enclose bright, spherical, spore-like granules of uniform size, arranged in linear series, and separated from one another by hyaline intervals. After having been stained with methyl blue, fuchsin or magenta, and then washed in nitric acid ten per cent., they retain the original dye, and are thus distinguished from any putrefactive or other bacilli.[1]

(2) This organism is only capable of growth and multiplication under culture in blood serum, or animal broth, at a constant temperature of 30° centigrade. It is of comparatively (to other bacteria) slow growth, and is unable to continue its development in decomposing fluids in the presence of more rapidly growing putrefactive bacteria. (Koch.)

(3) All the conditions essential for the development of the bacillus, are, so far as its life history is known, alone to be found naturally in the animal body.

(4) The bacillus is however, of very tenacious vitality, and will preserve its virulence and capacity for development, for six weeks or longer in decomposing sputum, for six months or longer in the dry state.

(5) If a minute portion of bacillus-containing matter be placed upon a neutral culture-surface, and allowed to germinate, and if a fragment of the product of germination be similarly cultivated on a fresh surface, and so on for many generations, all foreign germs being excluded, the last product, if inoculated into an animal, will be as potent in producing tuberculosis as the first.

(6) The bacilli, whether derived from free cultivation or from tubercle, if intimately diffused in water, and scattered in the form of spray through an atmosphere in which animals are placed so that they inhale it, will produce tuberculosis in them.

The following is a summary of our knowledge respecting the distribution of the bacilli in the lesions of phthisis.

(7) In the sputa of all cases of well-marked phthisis, the bacilli are to be found. (Plate II., fig. 2.)

(8) In cavities in the lungs of tubercular or caseous pneumonic, *i.e.*,

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[1] Klein and Gibbes on the relation of tubercle-bacilli to artificial tuberculosis. Supplement to the 13th Annual Report of the Local Government Board, 1883-84, p. 177.

of phthisical origin, whether large or minute, bacilli are invariably to be found. (Plate I., fig. 2.)

(9) In caseous and catarrhal pneumonic consolidations of the lung, excepting in the immediate neighborhood of cavities, large or minute, bacilli are sparse and rather difficult to find, large fields of sections may be traversed without discovering them; yet this material is virulent in producing tubercle when inoculated.[1]

(10) In the granulations of miliary tuberculosis, bacilli are very generally but not invariably to be found, and often only in small numbers.[2] In their most recent researches upon the artificial inoculation of guinea-pigs with tuberculous (bacilli-containing) sputum, Drs. Klein and Gibbes found that the tubercular lesions contained but few and in many instances no bacilli.[3]

(11) The results of inoculations made with dry bacillus culture by Koch and many others with the most minute precautions, have with much reason been accepted as proving the organism to be *per se* the virus of tubercle. These results have not altogether escaped challenge, however, and another yet possible fallacy is indicated by the fate of the very analogous cultivation experiments of Sattler with the jequirity bacillus. Dr. Sattler observed that in an infusion of the seeds of the *abrus precatorius* or jequirity plant—which when applied to the eye produces severe conjunctivitis—after a certain time bacilli appeared, which after cultivation on meat-serum, gelatine, etc., for many generations would produce in a rabbit severe ophthalmia, yielding secretions containing the bacillus. Klein confirmed these experiments in every particular, but he observed further that if the infusions of jequirity containing the specific bacilli were heated to the boiling point for half or one minute, the bacilli were not killed but could be cultivated on meat jelly as before. This short process of boiling had, however, rendered the jequirity infusion inert, and the bacilli although their vitality was unimpaired, had lost their virulent properties. It was obvious that a portion of chemical poison from jequirity adhered to the organisms of Sattler's dry culture experiments.[4] This poisonous principle had indeed, prior to Dr. Klein's

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[1] The most extended examination into the distribution of bacilli in the lesions of phthisis, is recorded in Dr. Percy Kidd's paper, read before the Medico-Chirurgical Society this year, see Transactions, vol. lxviii.

[2] In bovine tuberculosis the tubercle invariably contains bacilli in considerable numbers; the bacilli are smaller than those of human tubercle, and are much more generally contained within the cells of the textures affected. Klein and Gibbes, loc. cit., p. 178.

[3] Loc. cit., p. 182-183. Dr. Gibbes has been kind enough to show me some of his specimens from these animals of typical early stage tuberculosis of liver and lung, in which no bacilli are to be found. See also Kidd, loc. cit., p. 107.

[4] Pathological researches, by Dr. Klein, 13th Report Local Government Board, p. 147.

experiments, been discovered, and separated as an amorphous solid, "abrin," by Drs. Warden and Waddell in Calcutta.[1] In the recent discussion on Dr. Kidd's paper, Dr. Creighton expressed himself as not entirely satisfied that dry culture experiments were free from fallacy of this kind.[2]

It cannot, then, be said that the position of the tubercle-bacillus with regard to the etiology of phthisis is as yet established, although so intimate and exclusive is its association with the lesions of that disease that by its recognition in excreta or expectoration, we obtain a valuable criterion for diagnosis in obscure cases.

The etiological question at issue is of the widest possible importance. The doubtful position of the bacillus does not affect established facts with regard to the infectiousness of tubercle and its contagiousness under artificial and possibly under certain natural conditions, but it essentially affects our views as to whether phthisis is or is not a zymotic disease, and darkens counsel with regard to preventive measures.

We cannot say with phthisis as with the more definite zymotic diseases that we have health on the one hand and a specific organism on the other; that when we observe a man sickening with phthisis, the tubercle parasite is already in possession of him; and that we might hope by exterminating the bacillus to eliminate phthisis from our list of diseases. The characteristic lesions of phthisis are brought about by many causes, and furnish a soil upon which the tubercle-bacillus will readily grow. Epiphytic in nature, concomitant in time, neither the seed nor the fruit of the disease, it must nevertheless be allowed that the tubercle-bacillus takes an important part in the extension and conveyance of tubercular lesions.

Notwithstanding the apparently insurmountable antagonism between those who adhere to the essentially bacillous nature of phthisis and those who do not, there is a neutral ground where the two views meet, and where they may perhaps ultimately agree. Even Koch himself believes that certain pathological changes are, if not necessary, at least highly favorable to the reception of the germ. "We have no doubt," observes Dr. Weber,[3] "that the bacillus is intimately associated with phthisis, but the exact relations appear to require still further elucidation. It is, for instance, not quite clear why the tubercle-bacillus thrives in some persons and not in others; or why, in some persons, it thrives at one time and not at another. It seems to be an acknowledged fact that some micro-organisms do not grow in living tissues of a living animal. Dr. Klein states this with regard to the septic and zymogenic organisms properly so-called; and explains their occurrence in diseased tissues during the life of the subject, by assuming that these tissues had become practically dead before the organisms could grow in them. By analogy, the question arises whether the tubercle-bacillus settles and grows in healthy living tissues of the body or only in pathologically altered tissues. We know that it thrives in the bodies of most warm-blooded animals when inoculated, but this does not prove that it will find a nidus in a healthy tissue when merely brought into contact with it by the surrounding air."

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[1] The non-bacillar nature of abrus poison, by C. J. H. Warden and L. A. Waddell, Calcutta, 1884.

[2] See remarks, Proceedings Royal Medico-Chirurgical Society, N. S., vol. i., p. 303.

[3] Croonian Lectures, 1885, Lect. I.

It is our duty to consider what are the conditions which bring about this "aptitude for the reception of the bacillus"—to use the language of the contagionists, an aptitude which in the opinion of others constitutes incipient phthisis.

Experimental observations have abundantly shown that tuberculosis may be produced by inoculation of phthisical products in animals. Clinical experience and *post-mortem* observation furnish evidence of the conveyance of tubercular matter from one part of a lung to another, setting up fresh grafts of disease; also of a more subtle conveyance of the tubercle poison from centres of disease through the medium of vessels and lymphatics to neighboring or distant parts of the lung or other organs. Nor are cases wholly wanting in which inoculation from one human being to another has proved effective.

Contagion.—There is much in the nature of phthisis, and in the bent of the recent researches above alluded to, to encourage a belief in the contagious nature of the malady, a belief that was strongly held in the last century, and which in quite recent times has again in some measure returned.

My own personal experience and observation convince me, that apart from artificial conditions—such as those brought about by experiment—and in the ordinary circumstances of life, phthisis is not an infectious malady.[1] Nevertheless cases have even been recorded which are sufficient to convince others to the contrary, and "it would be rash to say that in no conditions of human association, can the degree of concentration be reached which might induce tuberculosis in a healthy person."[2]

Climatic causes.—The only positive climatic influence made out in relation to phthisis is that dependent upon insufficient surface drainage. Towns, villages, hamlets, and houses situated at or near undrained localities, on heavy, impermeable soils, or on low-lying ground, and whose sites are consequently kept damp, have a much larger number and proportion of cases of consumption than those which are situated on dry or rocky ground, or on light, porous soils, where the redundant moisture can easily escape.[1]

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[1] Consult on this subject Dr. Bowditch, Is consumption ever contagious? Boston, 1864; Villemin, Études sur la Tuberculose, 1868; Budd, Lancet, Oct. 12th, 1867; Thomson, On Phthisis in Victoria; Burney Yeo, Contagiousness of Pulmonary Consumption, 1882; C. T. Williams, British Medical Journal, Sept. 30, 1882; R. D. Powell, loc. inf. cit.; Andrew, Lumleian Lectures, 1884; Collective Investigation Record, vol. i.

[2] On the Causative Relations of Phthisis, by the Author, British Medical Journal, Oct. 11th, 1884.

"Phthisis is invariably present in low and damp countries (Lancereaux),[2] Buchanan,[3] Bowditch,[4] and Middleton,[5] have established the relationship in this country and in America between phthisis mortality and wetness of soil, Buchanan, especially, having observed the converse, viz., the diminished prevalence of the disease as one of the results of improved surface drainage."

Phthisis most abounds in sub-tropical and temperate climates and within the lower ranges of elevation. M. Jourdanet maintains that the disease almost ceases to appear at the half distance between the sea-level and the snow-line for any given latitude.[6] Whilst these statements are generally speaking true with regard to the climatic distribution of phthisis they are by no means absolutely so, nor can it yet be said how far the prevalence of the disease within certain limits is due to climatic influences, for it must be observed that the regions of the earth included are those in which the insanitary social and commercial conditions of civilized life most prevail. It has been suggested that the prevalence of phthisis in low-lying and ill-drained situations may be explained by the presence of conditions favorable to the preservation of the tubercle-bacillus; but it must be recalled to mind on the one hand that the bacillus flourishes nowhere outside the living body, and on the other that its vitality is not destroyed within ordinary limits by external conditions.[7] The experiments of Miguel and Emmerich have demonstrated what would necessarily be inferred, viz., that the proportion of bacteria in the atmosphere bears a numerical relationship to density of population and to barometric states, being most abundant in crowded cities (55,000 in 10 cubic metres in Paris), but absent in high latitudes and comparative solitudes (at 4000 metres elevation). M. Miguel attributes the absence of bacteria in elevated regions (1) to lessened dust, (2) to lessened power of the diluted air to hold particles in suspension, (3) to lessened centres for production of bacteria.[1]

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[1] Seventh Annual Report of the Registrar-General for Scotland, p. xlviii.

[2] Distribution Géographique de la Phthisie Pulmonaire.

[3] Simon's Reports to the Privy Council, 1867, 1877.

[4] Bowditch. Consumption in New England and elsewhere, 2nd edit., Boston, 1868.

[5] Paper read before the British Association at Bath, 1864. Mr. Middleton appears to have regarded the foulness as well as humidity of atmosphere as effective in augmenting the phthisis rate, whilst Buchanan more strictly refers to wetness of soil.

[6] Quoted by Lancereaux, p. 30.

[7] De la Phthisie Bacillaire, Prof. Sée, 1884, pp. 19, 20, 74.

These observations are valuable in precisely demonstrating the relative freedom of different localities from putrefactive products. Tubercle-bacilli have as yet been but rarely observed in atmospheric examination, so rarely as to render their presence apart from the question of direct contagion, which involves close proximity and the association of other elements of the disease, of little importance.

"A fruitful source of phthisis is the tendency to catarrh of the respiratory mucous membrane," observes Dr. Hermann Weber,[2] and further on he points out how these catarrhs may lead to phthisis, viz:—(1) By producing numerous mucous abrasions upon which the bacillus can settle; (2) by weakening epithelial cells and their ciliary action, in favor of the parasite; (3) by rendering respirations more shallow to the same effect; (4) "by weakening the nutrition and energy of the whole system."

Amongst the more disputed conditions in connection with climate, it cannot escape observation that phthisis is nourished under those influences favorable to the prevalence of catarrhal and inflammatory affections of the air-passages—more especially bronchitis and pleurisy—that is, on cold and damp soils, with variable temperature; and, as we know, at those seasons of the year, the spring and autumn, when these conditions prevail. In high and especially in dry situations, catarrhs are much less prevalent, respiratory conditions are more robust and active, and the processes of nutrition go on more vigorously in those to whom such climates are favorable.

Dusty employments.—The effect of dust inhaled in the pursuit of various employments has been exhaustively shown by Dr. Greenhow in the *Public Health Reports* for 1860 and 1861. Mining, flax-industries, pottery work, tool grinding, lace-work, etc., were all examined into, and, all allowances being made for social causes above alluded to, that the inhalation of dust remained as the positive cause of the undue prevalence of phthisis in the industrial localities, was shown by the fact "that the high death-rate from lung-disease belonged, according to the occupation, to men or to women of the district; that it sometimes was nearly twice as high for the employed sex as for the unemployed sex; and that it only extended to both sexes when both were engaged in the occupation."[3]

It is a remarkable fact that in those cases which are most typical of such mechanical origin the disease is very generally and strikingly one-sided, nor is the right side very notably more prone to attack than the left. Where on the other hand the fibroid characters of the disease are less marked, and the constitutional proclivity more definite, the two lungs are from an early period involved.

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[1] Quoted in Prof. Sée's work, p. 80.

[2] Croonian Lectures, 1885, Lecture I.

[3] Simon, Third Report Med. Off. Privy Council, 1860, 1861, p. 34.

Social conditions.—An examination of both climatic and industrial conditions predisposing to phthisis, cannot fail to convince any one how largely they are mixed up with conditions that fall under the heading of social relations. Phthisis is essentially a scourge of what we call civilization. Its rarity amongst nomadic tribes and savage populations in all climates, its prevalence in littoral districts, in river sites, and in industrial as compared with agricultural localities, in townships and cities—all point to social rather than to climatic influences, as predominant in the cultivation of the disease. All the depressing conditions of life—anxiety, mental strain, disappointments, bad sanitation, over-crowding, debauchery—are concentrated at the centres of civilization. The weakly are helped to live; rickets, scrofula, syphilis, catarrhs prevail; the general tone of health is depressed; recovery from acute specific disease—measles, whooping-cough, and from inflammatory chest-diseases—is less complete; germs, putrefactive and other, are so rife, that special precautions against them are necessary to secure the healing of wounds. All that we know about phthisis would lead us to expect its prevalence under such conditions. And it is comforting to find statistical evidence that with improved sanitation, there has been an appreciable decline in the death-rates from phthisis.[1]

Constitutional liability.—Apart from all other etiological considerations of phthisis, the constitutional liability to the disease must be taken into account, this constitutional proclivity being most purely and strikingly manifested in *hereditary liability*, more complexly so in *acquired* liability. I do not maintain the first, and it would be absurd to argue the second, as being independent of the surrounding climatic and social conditions which I have already discussed; but it is of the utmost importance in order to gain a clear insight into these etiological factors, with a view to measures of prevention, that they should be considered separately.

I have elsewhere defined the constitution of a man as "his build, the integrity or otherwise of the tissues of which each part of his body is made up, and the wholesomeness or otherwise of the juices with which they are bathed; the sum of his vital force, his cell-quickening power which shall bear the call of judicious expenditure, for a long or but a brief period of time."[1] This material and dynamic constitution is born with the infant, developed during the period of growth, and maintained with waning completeness during the wear and tear of subsequent life. Hereditary constitutional defect means unsoundness of original construction with regard to some organ or tissue at birth. Acquired constitutional defect means that some part of the human mechanism has suffered deterioration from deficient supply of the needs of growth and function, through willful or involuntary exhaustion of vital powers, or from imperfect recovery from acute disease.

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[1] The death-rate has declined at every age with some insignificant exceptions in both sexes; the decline being for males 14, for females 22, per cent. For both sexes the decline was greatest (28 per cent.) between the ages of 15 and 20, when deaths may be considered, in an economic sense, the greatest loss......The decline has been not much less considerable among both males and females at all other ages below 25, varying from 18 to 26 per cent. The mortality of males above 35 has diminished very little, but that of females has fallen between 14 and 24 per cent. until the age of 75, after which there is a trifling rise. The lives saved amounted to 3,966 males, 6,806 females; not less than 2,885 of the former. and 4,233 of the latter, were of ages 15-35, that is, were among the most useful of the community.—Longstaff, "On the Decline in the English Death-rate." Journal of Statistical Society, June, 1884, p. 226.

Inherited tendency to phthisis.—It is generally agreed that family predisposition obtains in 48 per cent. of cases of phthisis, and that the hereditary influence preponderates in females in the proportion of 59 per cent. to 37 per cent. males. It has, however, been well pointed out by my colleague, Dr. R. Thompson, to whose able work[2] founded upon the *Brompton Hospital Records*, we owe the most recent information on the heredity of phthisis, that the discrepancy in this particular between the sexes is due not to the working of any law respecting heredity, but to the greater exposure of males, and the consequent increase in the prevalence of acquired cases amongst them. When the hereditary influence is watched through the offspring of consumptive parentage, it is seen that the numbers of the two sexes affected become nearly equal. Dr. Thompson comes to the same conclusion from another kind of calculation.[3] Thus out of 3000 male cases taken consecutively from the *Brompton Hospital Records*, he finds that 36 per cent. have a family history of phthisis, out of the same number of female cases 58 per cent. have such a family history. But taking the mean of the three estimates of the relative *liability* of males and females respectively to phthisis, given in the first *Brompton Hospital Report*, and by Dr. Pollock and Dr. Williams in their works he finds it to be in the proportion of 62 for the males, and 38 for the females, and there is a near equality between 36×62, and 58×38.

Attempts have been made to throw some doubt upon the reality of hereditary influence in the etiology of phthisis.[4] The reality of this influence, however, is only too certainly a matter of experience, and did it need demonstration, such may be found in the account of 80 families of consumptive parentage given by Dr. R. Thompson, of which there were born 385 children, of whom 194 became phthisical, and 37 died in childhood, leaving only 154 exempt.

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[1] Etiological Relations of Phthisis.

[2] The different aspects of family phthisis in relation especially to heredity and life assurance, by Reginald Thompson, M. D., 1884, p. 37.

[3] Loc. cit., p. 40.

[4] See Walshe, last edit., p. 461, and the same author in the Brit. and For. Med. Chir. Rev., January, 1849.

1. The effects of so-called hereditary influence in leading to phthisis, have been regarded as attributable to contagion from the parent subsequent to birth either directly or through the milk. If this were so it would obviously follow that maternal "inheritance" should be far more deadly and earlier manifested than paternal. For the child is from earliest infancy in more constant association with its mother, and infected milk supply of parental source can only come from the mother. Nothing of the kind is observed in practice, however; and statistics show (*a*) that the "influence of *paternal inheritance* is especially developed before 25 years of age, being loaded upon the period between 10 and 25, the acme of susceptibility being exhibited between 20 and 25;" but that (*b*) with regard to maternal inheritance "susceptibility is not marked before 15, but is especially loaded on the period from 15 to 25, the acme being reached between 20 and 25."

2. It is said that inheritance in part arises from transmission of the virus in the manner in which syphilis is transmitted, and manifests itself early in life in the form of scrofula or meningeal or peritoneal tubercle (See *loc. cit.*, p. 60). Careful observation of the manifestations of scrofula in the form of caseous glands or diseased bones, undoubtedly favors the view that they are predisposed to by constitutional states, but is also convincing of the fact that they are called forth by local injury or distal irritation; a scratch, a cut, an eczema, a carious tooth, an otorrhœa, a catarrh of the bronchi or bowel, begets gland irritation and caseation follows. The manifestations of syphilis have, on the other hand, in their independence of such exciting causes and in their symmetry and early developmental features, the special characters of a blood disease; of course in the many cases in which inheritance is not immediately from one or other parent, any comparison with syphilis is impossible.

3. A third hypothesis, which is at once in accord with previously held views, and those most recently advanced as to the nature of the disease, is that suggested rather than expressed by Dr. H. Weber in his recent lectures at the College of Physicians, viz., that phthisis is inherited as a predisposition, an inherent quality of soil favorable to the development of phthisis, but which yet can only be fertilized by the specific spores of that disease. This may of course be so; there are abundance of spores about, and it comes to be almost a mere matter of account whether the phthisis be attributed to the spores or to the morbid state of tissues prepared for their delectation. In its transmissibility from either parent, its occurrence consequent upon, or in anticipation of, declared disease in the parent, in its declared presence being not protective from, but predisposing to, future attacks, and in the fact of atavism being a frequent

and important characteristic, phthisis is, with regard to heredity, strikingly different from syphilis and zymotic diseases and strikingly in accord with insanity and trophic diseases. (Thompson.)

A careful and impartial examination of the cases which come before us cannot fail to convince that neglected catarrh is the most common exciting cause of phthisis, although there must for the present be some difference of opinion as to the connecting link between the consequences of simple catarrh and the onset of phthisis.

CHAPTER XVIII.

ON THE VARIETIES OF PHTHISIS.

IT was held by Laennec, and it has in recent times been vociferously maintained, that phthisis is one disease. It cannot, however, be questioned that the range of pathological varieties within the sphere of this single malady, is so great as to justify its sub-divisions for clinical purposes of prognosis and treatment into sub-groups and varieties. Without further apology, then, I shall endeavor to sketch the main features of the following types of the disease.

PNEUMONIC PHTHISIS.
- Alveolar catarrh.
- Catarrhal phthisis.
- Acute pneumonic phthisis. (*a*) Confluent form. (*b*) Disseminated form.
- Chronic pneumonic phthisis.
- Fibroid phthisis.

TUBERCULAR PHTHISIS.
- Acute.
- Chronic.
- Acute tuberculosis—rather an infective complication of phthisis and of some allied conditions than truly a variety of that disease.

Other varieties separable for clinical convenience:—
- Bronchi-ectatic phthisis.
- Diabetic phthisis.
- Dust (colliers', millers', etc.,) phthisis.
- Abdominal phthisis.
- Laryngeal phthisis.
- Syphilitic disease of the lung and syphilitic phthisis.

There are further to be considered separately, on the grounds of mere convenience two of the prominent consequences of phthisis, about which are grouped some of the most characteristic features of the disease, viz.:—

Lung excavation.
Hæmoptysis.

A third consequence, of rarer occurrence, viz., pneumo-thorax, has been already fully dealt with in a preceding chapter. (Chap. vii., p. 88).

PNEUMONIC PHTHISIS.

ALVEOLAR CATARRH.—Alveolar catarrh in the first and slightest degree, forms the connecting link between the prodromal catarrh of Niemeyer and catarrhal pneumonia. This condition is extremely common, and very readily overlooked, for the signs by which it is recognized are only faintly marked. It must be considered as really the first stage of phthisis—through which all cases of pneumonic phthisis, and therefore the majority of cases of pulmonary consumption pass.

Whether this catarrh be specific from the first, in the sense of having been determined by the reception and germination of the tubercle bacillus, is a question sufficiently discussed in the section on etiology, and which need not further embarrass us at present.

The pathology of this disease consists, as has been before intimated, in the proliferation of the epithelium of the air-cells by a catarrhal process of the most superficial kind. Tubercle, in the sense of miliary granulation, has nothing whatever to do with this process, which may, indeed, pass on to catarrhal pneumonia and the destruction of the lung without such tubercle taking any conspicuous part in it.

The subject of alveolar catarrh has always been previously depressed in health, through tardy convalescence from some other disease, bad living, mental anxiety, or overwork; he has had a persistent, though it may be a slight, cough, for a longer or shorter time, and has during that time been getting thinner. Professor Niemeyer, who has more than any other author urged the importance of the early detection and treatment of this condition, regarded the presence of pyrexia as the one symptom above all others significant of alveolar catarrh, and he also justly attached great importance to the occurrence of streaky hæmoptysis. The pyrexia may not amount to more than a slight evening rise of temperature, but it is attended with malaise and increased cough at night.

At this stage the physical signs are very slight, but in conjunction with the symptoms are sufficient for diagnosis; the respiration is weaker at one apex, the inspiration being wavy, or even jerking. There are usually a few ronchi present, which, if limited to that apex, are very significant, and, in addition, there is heard at the extreme summit of the lung (supra-clavicular or supra-spinous region) a peculiar crumpling sound at the moment of cough, which differs both in time and degree from the crepitant sound audible at a somewhat later stage with the first inspiration following a cough.

These physical signs, which are but very slight—and their recognition is of the more importance on this account—are those of a bronchial catarrh limited to one apex, associated with a decided imperfection of the respiratory murmur at that apex, which, when taken in conjunction with the symptoms—more particularly emaciation, quick pulse and evening pyrexia—afford unmistakable evidence of incipient phthisical disease, upon which we must advise most decidedy if we do not wish to see the patient pass beyond our control so far as positive cure is concerned.

There is no clinical line of demarcation to be drawn between this condition and the prodromal catarrh which precedes it, or the catarrhal pneumonia into which it is apt to pass; they shade imperceptibly into one another. I only specially refer to this as the earliest recognizable stage of phthisis.

CATARRHAL PHTHISIS.—The following case is illustrative of catarrhal pneumonia in an early stage and bears out some of the above remarks respecting the most common etiology of this form of consumption.

S.S., aged 29, a married woman engaged in domestic duties and suckling a child aged seven weeks, came under my notice as an out-patient in April, 1871. Her mother had died of consumption within two years of the patient's birth, and an elder sister had been affected with the disease in an early stage. She had enjoyed fair health until her first confinement, when she was with difficulty delivered of twins, only one of whom survived the birth. This child she suckled for eleven months, when she again became pregnant. Ever since her last confinement she had suffered from increasing debility, emaciation, and cough, and shortly before that time she had had slight hæmoptysis.

She was a tall, thin, anæmic woman, with the worn look so characteristic of over-lactation or rapid child-bearing; her large, heavy, pendulous breasts, marbled with large veins, increased by contrast the general flatness and narrowed antero-posterior diameter of the chest. There was no local flattening, however, at either apex, and the respiratory movements though generally deficient were not more so at one apex than at the other. On percussion over the summit of the left lung the resonance was somewhat less than on the opposite side; the respiratory sounds there were harsh, and accompanied by some moist crepitation which extended to the second rib. The respiratory murmur elsewhere was of fairly good quality, but somewhat feeble. The main symptoms complained of were troublesome cough and yellow expectoration, shortness of breath, general weakness, and giddiness in the head. The pulse was quick and weak, the appetite indifferent, but digestion fairly good.

The case was regarded as one of catarrhal-pneumonic phthisis in an early stage, the disease being limited to the left apex, and supervening upon the exhausting effects of more than thirty months' continuance, alternately, of gestation and lactation. She was directed immediately and completely to wean the child, to take abundance of appropriate food, with a moderate amount of beer. Some counter-irritation was applied at the left apex, and cod-liver oil and steel wine administered, with some sedative lozenges for the night cough.

On again examining the chest a month later, the moist sounds were no longer audible with ordinary respiration, but a few crackles were heard after cough. There was slight flattening at the left apex, which became more obvious on deep inspiration; the respiratory sounds were feeble there, while on the opposite side they were more developed, and on percussion the line of resonance of the right lung extended a little to the left of the mid-sternal line. There was no evidence of a cavity at the left apex, and no extension of the disease below. The health of the woman was, though improved, by no means restored; she was still anæmic and thin. Her cough was troublesome, especially in the morning, and expectoration difficult—the efforts of coughing often causing vomiting at that time. She had neglected to completely wean her child. The pulse was quiet but weak; the appetite improved. Three weeks later she had very greatly improved in health and strength; some color had returned to the cheeks, and she was gaining flesh rapidly.

This case would be included under the heading of pneumonic phthisis, by Addison; catarrhal-pneumonia, by Niemeyer, Hérard and Cornil; an epithelial pneumonia by Andrew Clark. By Laënnec it would be termed local pulmonary tuberculosis, and by some modern writers it would be spoken of as early bacillary phthisis. Bacilli were not recognized at the time this case occurred, but we know from abundant experience that they are to be found on sufficiently diligent search in all cases in which the signs of softening are present.

It is fair to assume that, had this patient been in happier circumstances, had her health not been depressed by the development, at her expense, of three infants and the maintenance of two of them, while she herself was doubtless not in the enjoyment of nutritious food in any great abundance, had she sought advice earlier (and taken it), she would never have become phthisical—her hereditary proneness to consumption might have remained a mere latent tendency. Had there been any family tendency to insanity, it is quite possible that the same evil conditions might have caused her to be afflicted with some form of puerperal mania. "It is true that privation, excess, errors in habits of life, the sedentary occupations, the pernicious influence of certain trades, grief, anxiety, and the other wasters of vital powers, will not suffice to induce consumption in all, or even in the greater proportion of individuals; for these agents so universally prevalent are part of the daily lot, or of the daily errors, of many more than fall victims to consumption. But it is also true that, if to any or all of these conditions that of inherited tendency to phthisis be superadded, very few indeed escape the disease."[1] This remark is well borne out by the above, amid numberless other cases which must be familiar to physicians.

The points about the case which rendered the prognosis a favorable one, with certain reservations, were:—1. The obvious and very sufficient determining cause. 2. The limitation of the disease to one apex. 3. The presence of considerable crepitation and some dullness, without any local flattening or marked difference in expansion. 4. The absence of fever at the time of coming under observation.

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[1] Dr. Pollock, Elements of Prognosis in Consumption, p. 340.

1. If the circumstances of the patient admitted of complete rest from the cares and anxieties of her position of life, and change to a purer air, there would scarcely have been a doubt as to the prognosis. Without these advantages the disease ceased to extend, the secretion sounds dried up, and flattening appeared with some corresponding development of the opposite lung.

2. In addition to the evidence of the integrity of the opposite lung, the encroachment of its margin towards the diseased side should always be anxiously looked for; it can be readily made out by percussion, and when the disease is one-sided, it precedes, often by a long interval, any decided apex flattening, and is always a sign of good augury in prognosis.

3. The late appearance of flattening—coincidently, that is to say, with the lessening and disappearance of moist sounds, which have been considerable—is an important sign of arrest of the disease, in contradistinction, to flattening which comes on coincidently with an advance in the other physical signs, and which may be due, therefore, to sheer loss of lung substance, or to the presence of the indurative form of disease, chronic pulmonary tuberculization, which, though of chronic course, is yet one of the most intractable of lung affections. Flattening must then only be considered in conjunction with other signs, and with especial regard to the period of its appearance. In the case before us it signified, together with the other signs, pulmonary collapse, with perhaps a few shrunken nodules, adhesion and some thickening of the pleura.

I believe that most cases of "cured" early-stage phthisis are of the kind above related. The cure may remain permanent, but one must always bear in mind that catarrhal pneumonia is one of those diseases that are peculiarly prone to recur, resembling acute rheumatism, tonsilitis and some other diseases in this respect. The delicacy of the lungs, inherited or acquired, which has led to the first attack remains, nay, is increased by that attack. Hence the previous health history of the patient helps us much in the prognosis in each individual case.

Those cases in which the pulmonary delicacy is distinctly inherited are the least hopeful, those again in which the attack has been most distinctly led up to by adverse conditions of a definite and remedial kind are the most favorable. Of course in each case the extent of lung involved and the intensity of the disease, to be ascertained only by physical examination, must, as already pointed out, most importantly enter into the question as to prognosis.

The patient, whose case is above related, after some months had a second attack, resulting in extension of disease on the same side, involvement of the opposite lung, and a fatal termination.

In January, 1877, in the course of some clinical lectures delivered at the Brompton Hospital,[1] I related the following case in illustration of the rapid formation and cicatrization of a cavity at the right apex and the complete clearing up of all signs and symptoms.

A. W., a pale, scrofulous-looking girl, who was in attendance at the time of lecture, had been under my observation at the hospital since March, 1872, when, at the age of fifteen, she first came to me as an out-patient. She had then a somewhat loud cough, which had troubled her all the winter, but there were no other definite symptoms and no discoverable pulmonary signs. Her family history was good, but she had been delicate since an attack of measles in childhood. She got well on ordinary treatment, but returned in October, 1874, and again improved, but was ailing during the winter. In the spring of 1875, her principal complaint was of a painful affection of the left breast; she still had no definite pulmonary signs or symptoms beyond general delicacy and slight cough.

In June, she had whooping-cough rather severely; and six weeks after she still whooped with the cough, which was attended also with thick difficult expectoration. On again examining her chest I was somewhat surprised to find dullness with very marked cavernous breathing and some gurgling below the right clavicle; moist crepitant râles were also rather sparsely scattered over the posterior base. Fever and hectic symptoms were now marked. The prognosis appeared grave, and I recommended her to obtain an in-patient's letter. She did not come into the hospital, however, until January; meanwhile she improved as an out-patient. In October the sounds were noted as being quite dry, and on her admission into my ward in January, I examined her chest with great care, and did so repeatedly afterwards, but I could never discover more than some harshness and feebleness of breathing at the right apex.

I take it that this was a case of solitary *caseous abscess* at the right apex, which cleared out and cicatrized. Such cases are, however, unfortunately exceptional; this is the only one of the kind of which I have a definite and certain note. Other similar cases have, however, been reported, and *post-mortem* observation of cicatrices in the lungs leave little doubt that they occur more frequently than positive clinical experience would indicate; but they are rarely single and uncomplicated, as in this case.

The patient A. W. remained well after leaving the Hospital in 1877, and early in 1883 was married. In the course of that year she had a miscarriage and suffered much from menorrhagia afterwards. In May, 1885, she again came under my observation with cough especially during the night and in the morning, and some loss of flesh. She was nursing entirely a child eight months old. At the right apex, the seat of former mischief, there was slight dullness and some deep crackling and catarrhal râles but no evidence of a cavity. A few bronchitic râles were scattered over the chest on both sides. The breath-sounds below the left apex were not quite satisfactory.

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[1] Lecture ii., Lancet, Jan. 27th, 1877.

It is very common to meet with cases in which the second attack does not affect the same part of the lung as the first, but the opposite apex or some other portion of the same lung.

Acute Pneumonic Phthisis.—(1) *Confluent form.*—In other cases of phthisis of more acute character, still as a rule one-sided in the initial attack, the consolidation is more extensive and more dense, the disease being more "massed" in the affected lobe, occupying perhaps its entire extent.

This form of the disease is very apt to be confounded with croupous pneumonia of the apex—it very rarely occurs at the base of the lung—the signs of consolidation being uniform over an extended area; bronchial breath-sound and crepitant râles, at first fine, then coarser, are heard, and the voice sound is bronchophonic. The onset of the disease is abrupt and stormy, with pain in the chest, short cough, scanty expectoration, sometimes hæmoptysis, with raised temperature, rapid pulse, and frequent respirations.

The croupous element does indeed enter into the formation of many of these massed consolidations of the lung and it may be only after its resolution that the true nature of the case is unmasked with the slower decay and softening of those centres which have undergone caseation.

In the distinction of these cases, from the not uncommon instances of true croupous pneumonia of the apex the following considerations are important.

1. The temperature although maintained above the normal has even from the first a more fluctuating range than that of pneumonia. The tongue is as a rule moister and less typically coated than in pneumonia, and hectic sweatings very soon become marked.

2. No critical fall of temperature corresponds with, or follows upon, the appearance of moist sounds.

3. The hæmoptysis if present is decided, not a mere rust color of the sputum. The sputum is more abundant and from the first, more or less purulent, containing bacilli (Plate II., Fig. 1) and alveolar elastic tissue (Fig. 18).

4. As the case proceeds amid the moist crepitant râles, which cannot be distinguished from those of resolving pneumonia, larger clicks are heard in several centres, which become more liquid and characteristic of pulmonary softening.

5. The breath-sound, which had become more or less completely masked by the numerous râles, again appears in patches corresponding

with the large clicking râles and assumes a hollow tubular quality. The voice-sound over these areas becomes of a nasal, snuffling quality and finally there is distinct pectoriloquy.

6. The rapid emaciation and markedly hectic temperature of the patient, his profuse sweatings, and the increasingly copious and purulent expectoration, within a few weeks put aside all possible error of diagnosis.

Cases of caseous phthisis by no means all exhibit at the first onset the stormy symptoms above sketched. But in all cases the physical signs rapidly extend, until a more or less considerable portion of one lung is involved, before the signs of breaking down into cavities are manifested.

In persons of lymphatic temperament, who are the more common subjects of this form of phthisis, the fever may not be sufficiently violent, nor the other symptoms urgent enough to prostrate them in bed; but remittent fever, cough, expectoration, and hectic sweatings are invariably present and sufficiently manifest to excite alarm in parents and friends.

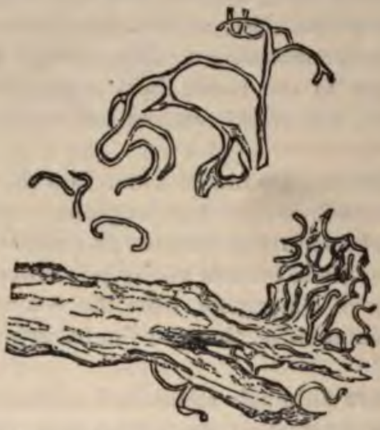

FIG. 18.—Elastic tissue and fragment of small vessel, from expectoration of patient with rapidly forming cavities.—Drawn by Dr. Sydney Coupland. × 200.

It is remarkable, however, to observe the rounded contour of features and the relative plumpness of figure maintained by patients even far on in this form of the malady, which is certainly most common in young girls and women. The features are usually pale with anæmic mucous membranes, but in the younger subjects the cheeks may be well-colored. The chest formation is usually fairly good, and in common with the rest of the frame well-covered with a layer of adipose tissue. The muscular system is, however, poorly developed and languidly innervated, the pulse feeble, and the nervous system wanting in tone.

I have pointed out that in the form of phthisis now under consideration there is a tendency to arrest. This is brought about:—

) By elimination of the caseous products.

(2) By cicatricial contraction of the cavities thus formed.

(3) By compensatory development of the opposite lung.

It is a mistake commonly and naturally enough made by the inexperienced observer, on having revealed to him through his stethoscope the signs of breaking down of pulmonary consolidation in one or more centres, to infer that a further step has been advanced towards a hopeless prognosis in the case before him. In a sense, however, and fully admitting the gravity of the case at the first moment of diagnosis, these phenomena of softening are equally essential and important steps towards amendment. Without them it is impossible for healing changes to ensue.

At the same time this process of elimination is attended with dangers of its own which have to be reckoned with.

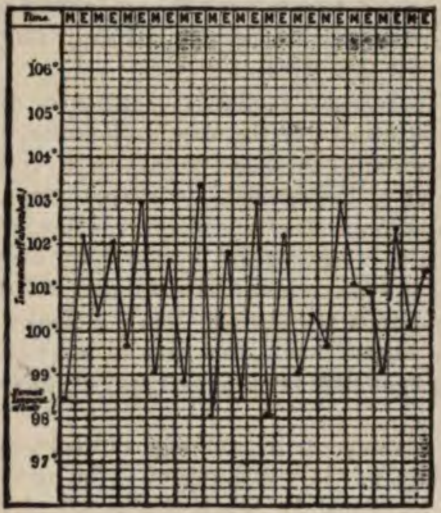

FIG. 19.—Annie T.—Fluctuating hectic temperature—Acute pneumonic phthisis—Cavities rapidly forming.

(*a*) Suppurative fever necessarily attends the process. A fluctuating hectic temperature, night sweats, cough, and more or less profuse expectoration, containing elastic tissue and bacilli; hæmoptysis is rarely present to any great extent. The character of temperature chart marking this period of phthisis is precisely the same as that of an empyema, or an imperfectly drained abscess in any other part of the body, or the more brief hectic period of typhoid fever. Figs. 19 and 20.

(*b*) There is throughout this period a danger of the opposite lung becoming involved in separate centres, from inhalation of specific morbid products in course of expectoration. Whilst the possibility of secondary tuberculosis at this stage is not to be forgotten, it is, so far as I have observed, a complication of much more rare occurrence than in other stages.

(*c*) The patient tends to become more and more exhausted, and to lose weight throughout this period, and in severe cases constitutional resources are seriously taxed by a downward course of from six weeks to three or four months.

During the process of elimination, cavities gradually become manifested to auscultation, and perhaps increase by coalescence to form one or two amphoric areas. Some shrinking of lung from loss of substance may be observed, the heart's surface being uncovered more or less to right or left according to the side affected.

Coincidently, however, with these alarming signs of lung destruction, it may in a goodly number of cases be observed, (*a*) that the opposite lung has not become affected to any material extent, and that the limits of the disease on the affected side have not extended but have perhaps even retrenched. It is necessary here to warn the auscultator against mistaking morbid sounds heard on the healthy side but really consonated from the diseased side, for signs of new disease.[1]

(*b*) Before the subsidence of hectic phenomena, careful percussion and auscultation will reveal that the margin of healthy lung is encroaching upon the sternum until the whole sternal region is thus occupied.

(*c*) The flattening of the affected side becomes more marked, and the area of cardiac pulsation more extended towards the diseased side, whilst the cardiac surface is being covered by the lung advancing from the healthy side. The percussion note becomes duller and more wooden, and the hollow breath-sounds are less and less abundantly accompanied by râles. It is to be further noted, especially in left-sided cases, that the morbid pulmonary signs shift towards the upper axillary and infra-spinous regions.

(*d*) The range of temperature gradually contracts, and this restriction may often be observed not only with regard to the high, but also the low register, for it is very usual for the hectic period of this disease to be marked by sub-normal points in alternation with pyrexial peaks (see fig. 20). The other phenomena of hectic abate, the patient regains appetite and flesh, and in favorable cases and under favorable circumstances the cough and expectoration may cease, the cavities contract and convalescence be fairly established.

(*e*) In left-sided cases when the lung has considerably contracted and all pulmonary signs have become quiet, the patient frequently complains much of palpitation and occasional faintness and ready disturbance of the heart's action. These symptoms are due to the organ being somewhat displaced, its anterior surface being unprotected by lung in contact with the depressed parietes, it being further bounded on the left by a thickened lung instead of its normal elastic cushion of support.

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[1] This fallacy has recently been pointed out by Dr. Skerritt in a paper On the Conduction of Physical Signs in Diseases of the Lungs, Brit. Med. Jour. 1884, vol. ii., page 1005. See also Chap. X., pp. 130, 131.

(2) *Disseminated form.*—In other cases, of happily rare occurrence, acute phthisis attacks both lungs, commencing simultaneously in many centres. This form of the disease bears the same relationship to the preceding that disseminated bears to confluent, broncho-pneumonia. It corresponds with the *florid phthisis* of some authors, the *phthisie galopante* of the French. *Acute disseminated pneumonic phthisis* is the most descriptive term for the disease. For, whatever views different writers may hold as to its origin or otherwise in tubercle, the rapid destruction of lung texture is unquestionably the result of inflammatory action. The symptoms are not essentially different from those of the more ordinary form of acute phthisis, but the dyspnœa is more urgent and the progress to a fatal issue rapid, and, as a rule, unbroken.

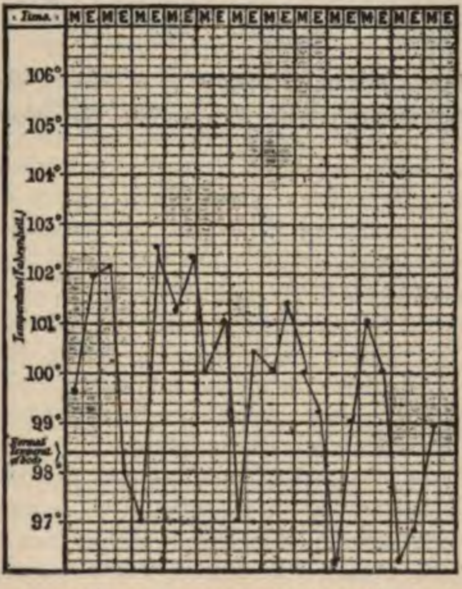

FIG. 20.—Marked hectic type of temperature attending the rapid softening and elimination of extensive caseous consolidation. Lucy B., late period of acute phthisis.

The flushed face, bright eyes and alert mind contrast with the apathy, pallor and prostration of acute tuberculosis, and the physical signs at first of acute bronchial catarrh with the rapid development of numerous centres of crepitation, softening and excavation, are equally characteristic. The rapid breaking down of the broncho-pneumonic centres occurring first at the apices, is again a characteristic feature, distinguishing this from mor simple forms of broncho-pneumonia.

Post mortem the lungs are found to present numerous areas of greyishpink granular consolidation, with yellow caseous centres broken down into small cavities communicating widely with enlarged, more or less eroded and acutely inflamed bronchial tubes; no miliary granulations of tubercle are to be seen, although the smaller, yellow centres may at first sight resemble them.

Except on grounds of clinical accuracy, the distinction between acute disseminated phthisis and acute tuberculosis is of little importance, for the *prognosis* in both is about equally fatal within a short period of from four weeks to two or three months.

The high and fluctuating temperature, hectic sweatings, purulent, sometimes blood-stained, and soon nummulated sputa containing elastic tissue and bacilli, in association with the physical signs, will render it impossible, except in the earliest stage, to confound this disease with acute bronchitis.

An inquiry into the family history of a case of acute disseminated phthisis will always elicit evidence of a decided phthisical taint.

CHAPTER XIX.

CHRONIC PNEUMONIC PHTHISIS—FIBROID PHTHISIS.

THE following case exemplifies fairly well the transition stages between chronic pneumonic and fibroid phthisis, a transition pathologically very easy, and clinically often to be observed.

John B., aged 29, a butcher's assistant, came under my notice in March, 1871. He was a broad-chested, powerfully made man, of medium height and florid complexion. He had led a rough but sober life, having followed his present business, which included the slaughtering of animals for some years in Australia, and had enjoyed excellent health until shortly before Christmas, when, after getting wet, he caught a severe cold, which was followed by a cough, which had since increased, uninfluenced by treatment. Up to and at the time of his attendance, he was still following his employment, but he now did so with difficulty, complaining of his cough and of increasing weakness with decided emaciation. His father had died of consumption, brought on subsequent to the patient's birth by intemperance; there was no other hereditary tendency to the disease.

The chest, as before said, was broad and well-formed, without flattening or obvious impairment of expansion. The heart's apex beat in the natural situation. At the left clavicular and sub-clavicular region the percussion note was dull, the dullness extending to the fourth rib; posteriorly, the resonance was defective at the left supra-spinous fossa. Scattered over the dull regions there was coarse crepitation, mingled with a still larger humid crackle. These moist sounds were abundant, and masked to a great extent the respiratory murmur, which was decidedly harsh, but not distinctly bronchial. Its vesicular quality became gradually restored as the stethoscope was passed downwards. At the posterior base there were some scattered sibilant râles. On the right side the percussion note was good, and the breath-sounds were natural.

The sequence of events in this case appear to have been a more or less general bronchial catarrh, subsequently localized at the left apex, extending thence into the alveoli, and there producing catarrhal pneumonia; yet the man continued his daily work, though constantly losing strength, for three or four months, during which time the catarrhal process ran on insidiously to a more deeply inflammatory degeneration of the alveolar walls. The disease had been acquired by exposure, the family tendency being slight, and the build of the chest not that of inherited tendency to phthisis. The physical signs at the present stage showed consolidated lobules of blocked alveoli, which were softening with varying degrees of rapidity; the coarse crepitation answering to the redux crepitation of pneumonia, the larger click being due to more profound destruction of tissue (softening). On the occasion of his first visit the pulse was quick and the tongue red, and although there was no elevation of temperature at the moment, it is probable that it rose slightly towards evening.

The patient was treated with an alkaline mixture containing small doses of iodide of potassium, and with cod-liver oil. The next note of importance was taken on April 27, when the expansion of the left side of the chest was found to be decidedly impaired, the dullness had increased in hardness but not in extent, and was very marked, especially between the left margin of the sternum and the mid-clavicular line.

In the space marked out by these two vertical lines (left sternal and mid-clavicular), the respiration was extremely feeble, and not attended with any râles; the heart's impulse was diffused to the second interspace, though the apex was only half an inch higher than natural. To the left, again, of the mid-clavicular line, the respiration was still feeble, and the râles much diminished, the dullness being somewhat greater than before. At the apex posteriorly there was bronchial respiration and imperfect pectoriloquy the bronchial râles at the base had cleared up. The resonance of the right lung extended to the left margin of the sternum.

These signs showed:—1. That the disease had not extended; on the contrary, the signs of bronchial irritation at the base had cleared up.

2. A wasting of the parenchyma of the lung had taken place; degeneration, absorption, and expectoration had removed the morbid contents of the alveoli, and some of the lung tissue itself, leaving, perhaps, at the apex a small cavity; the general result, however, being collapse and agglutination of air-cells. Hence a considerable reduction in the bulk of the lung and the retraction of its anterior margin *from the median line;* so that between the left sternal line and a line drawn from the point of junction of the inner and middle third of the clavicle to the apex of the heart, there was probably, at this date, no lung at all.

3. An encroachment of the enlarging right lung, a slight shifting of the heart to the left, and a flattening of the chest wall to make up for the lost space. The flattening was, however, as yet very slight, and not noticeable until the patient drew a breath. The man had powerful parietes, and in such cases the displacement of heart and encroachment of the opposite lung precede, often for a long time, any obvious flattening.

It was remarkable with what rapidity these changes were taking place, and there can be no doubt that the connective tissue of the bronchial and peri-vascular and pleural sheaths was undergoing rapid development, and that the case was, at this date, not merely one of catarrhal pneumonia, which had subsided after having caused a certain loss of lung substance, but that an interstitial pneumonia was proceeding; the case had changed its type to one of peri-bronchial, or rather, pulmonary fibrosis; it had lapsed into one variety of fibroid phthisis. That the disease was not yet arrested seemed probable from the patient still losing slightly in weight and becoming more anæmic; but it had clearly become limited.

During the next month he lost two pounds in weight. He was during this time taking an acid preparation of iron, with a little quinine, and the oil. Notwithstanding this slight loss of weight, he had improved generally; cough and expectoration had diminished, and he felt stronger. On June 8, he was still better, and had gained one pound since last report. He had very little cough; all moist sounds had disappeared except a slight pleuritic râle on cough at the outer side and a little above the left nipple. He steadily improved up to August, 1871, when I lost sight of him.

FIBROID PHTHISIS.

The term "fibroid phthisis" has been productive of much discussion. Originally introduced by Sir Andrew Clark as "embracing all those cases, whether local or constitutional, which are anatomically characterized by the presence, in a contracted and indurated lung traversed by more or less dilated bronchi, of fibroid tissue, and of tough fibrogenous substance, together with cheesy deposits or consolidations, and usually small cavities, commonly found about the middle and lower parts of the affected organ,"[1] the term is such a neat, concise, and clinically useful one, that it has been very generally accepted with some reservations as regards the strict pathology of the disease so designated.

The prominent symptoms and signs by which cases of fibroid phthisis are distinguished, are:—increasing contraction and immobility of the affected side, traction of organs to that side, deadened percussion-note and weakened breath-sounds of more or less bronchial quality, at parts intensely bronchial or cavernous; breathlessness, dragging pains, paroxysmal cough, occasional hectic, but general absence of fever; very chronic progress, long continued one-sidedness of the disease, and correspondingly slow failure of nutrition. Such symptoms and signs bring the cases within the definition of phthisis, but phthisis of a special kind.

The conditions presented *post mortem* are those of a contracted, toughened, indurated, and usually pigmented lung, surrounded by a greatly thickened adherent pleura, containing one or more rigid, dense-walled cavities, dilated bronchi, and cheesy encapsuled nodules.

On minute examination, this condition of lung is found to have been produced by a growth of two kinds pervading it. 1. Connective tissue proliferation, resulting in the formation of bands and processes of *fibrous* tissue, derived from the sheaths of vessels and bronchi, and the subpleural and inter-lobular tissue of the lung. 2. A more important nuclear growth leading to the formation of broad tracts of *fibroid* tissue, thickening the walls of the alveoli, compressing, and finally effacing them, unless they have been previously stuffed with their own catarrhal products; this fibroid structure being very possibly derived from an overgrowth of the lymphatic elements normally pervading the lung.

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[1] Clinical Transactions, vol. i., p. 188.

The products of these two processes become intimately mingled, but it is the latter which is the phthisical element in the disease, for mere connective tissue growth does not lead to organic destruction of the lung. This is also the element which specially gives to the disease its peculiar clinical features, and renders the name "fibroid phthisis" applicable to it.

The clinical application of the term "fibroid" thus corresponds with the use of other adjective terms—"catarrhal," "tubercular," etc., in describing the *predominant* character of the variety of phthisis thus distinguished; for in all cases of chronic phthisis the morbid processes are of more or less mixed character.

Fibroid phthisis is in the great majority of instances, so far as my own experience informs me, of a truly secondary nature, supervening upon some more or less acute inflammatory affection of the lung, whether lobar or lobular, catarrhal, or tubercular pneumonia. It is very doubtful whether pleurisy or bronchitis alone can give rise to it without the intervention of lobular pneumonia or tubercle. Local injury or pulmonary abscess may form the starting point of the disease, but are not alone sufficient to cause any extensive fibroid invasion of the lung beyond their own immediate limits.

Numerous examples may be found of this somewhat inclusive disease, ranging from the most typical cases to those which are indistinguishable from ordinary chronic phthisis. I have described a case of catarrhal-pneumonic phthisis, in which the transition into one of fibroid phthisis or pulmonary fibrosis was traced. It would not be difficult to find examples in which the reverse takes place, the clinical characters of fibroid phthisis being gradually changed by subsequent pneumonic processes, and all the features of the special variety becoming merged in the diffuse pulmonary destruction. Thus in a boy, to whose case I alluded in a previous edition of my work, the disease began with catarrhal pneumonic destruction of a certain portion of lung, upon arrest of which the marked phenomenon of pulmonary fibrosis supervened, and finally again active destructive changes set in quite obscuring the fibroid characters of the disease and resulting in death from pneumo-thorax.

Cases of fibroid phthisis may be roughly divided for convenience of clinical exposition into three varieties:—1. Those in which the disease has its starting point at the apex of the lung, and proceeds downwards. 2. Those in which it commences at the base, and advances upwards. 3. Those in which the most marked signs are discoverable about the middle of the lung. Of the first variety the following case is a fair example:—

George P., a sawyer, aged 43, came first under my notice as an out-patient at the Brompton Hospital in August, 1868. He was a thin man, with dark hair, having no hereditary predisposition to lung disease, except that his father had suffered from "asthma." In the preceding January, having suffered from slight cough for years, he was laid up for six weeks with "inflammation of the right lung." Since that time the cough had been continuous, and three months ago had been attended with slight hæmoptysis. The cough was now paroxysmal, causing retching and often rejection of food; expectoration difficult, abundant, and of a pink tinge. He had got thinner lately. The digestive functions were fairly good; the pulse a little hurried; there was no fever present.

On inspecting the chest, cardiac pulsation was visible at the fourth right interspace, Fig. 21, c, to left of right nipple. This side was diminished in size and much restricted in movement, the intercostal spaces deepening with inspiration; while the left side expanded freely, with an uplifting movement of the shoulder. On careful examination, the apex of the heart was found a little to the left of the ensiform cartilage.

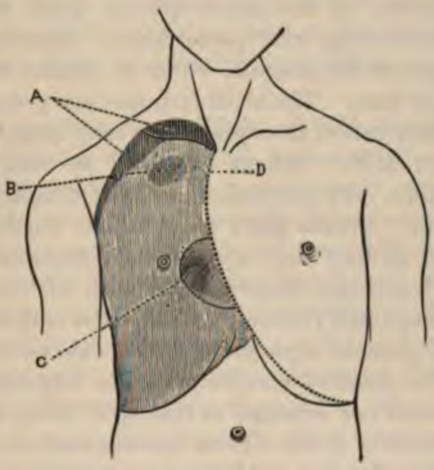

FIG. 21.

On percussion, the right side in the nipple-line anteriorly was dull to the second rib, comparatively resonant to the fourth, and below this point it was again toneless. To the left of the line of sterno-clavicular articulation, at the level of the second and third cartilages, there was good resonance continuous across the median line with that of the opposite side. The line of this resonance, D, sloped upwards to the episternal notch, being displaced in a downward direction by cardiac dullness at the fourth cartilage. Hepatic dullness barely reached the costal margin. There was dullness throughout the *axillary* region and *posteriorly* from apex to mid-scapula, the note having a tubular quality in this latter region. Below the mid-scapula there was fair resonance to the ninth rib, though less and harder than on the opposite side; the lower two or three inches on the right side gave a flat note on percussion.

The percussion-note over the whole left side, including the region of normal cardiac dullness and extending across the median line, as above indicated, was full and good in front and behind.

The auscultatory signs were in agreement with those of percussion. Above the clavicle on the right side, the respiration was amphoric, and dry; below the clavicle weak and bronchial to the base, with some rather large, moist rhonchus, friction, and bronchophony. At one spot corresponding with the second and third ribs, nipple-line, B, the breath-sound was of tracheal quality with scanty cavernous clicks and pectoriloquy. In the upper axillary region the respiration was amphoric, and the voice-sound pectoriloquous; in the supra-spinous fossa and inter-scapular regions, cavernous-blowing, with pectoriloquy. Blowing respiration extended to the angle of the scapula, where it became weaker and gradually annulled at the base. The vocal fremitus was generally increased on the right side. Respiration throughout left lung exaggerated vesicular.

February 27th, 1869.—Patient improved in flesh and appearance; stronger than before, but complains much of cough, and expectorates much pink phlegm. Breath short on exertion; cough causes retching, but does not bring up food now; appetite fair; digestion not very strong; bowels regular. Continues medicine (oil with nitro-muriatic acid and cinchona). Fingers noted (February 13th) to be clubbed.

The additional physical signs noted at this date were, a distinct, short, systolic bruit at the point of maximum cardiac impulse, not appreciably increased by pressure nor confined to that spot, being also audible at the apex. Measurements of chest:—From mid-sternum to nipple, right side, 4 inches; semi-circumference, 15¼ inches; expansion, ¼ inch. Left sterno-nipple measurement, 4¼ inches, semi-circumference, 16 inches; expansion, ½ inch.

We may, by way of summary, aided by a glance at the figure (reduced as accurately as possible from a sketch taken at the time upon an outline diagram), interpret the above detailed physical signs as indicating at this period a general induration of the right lung, with much contraction, its anterior margin having receded considerably from the median line, exposing the pericardium, and having also shrunk away from the upper surface of the liver. Its upper and a portion of its lower lobes were extensively excavated, the cavities being old, tolerably dry, and shrunken with the general contraction of the lung. The pleura (from the hardness of percussion, feebleness of breath-sound, and great fixity of walls) was probably greatly thickened. The liver was drawn up within the costal margin, and the heart considerably displaced to the right, its axis being, however, but little altered. A short systolic murmur was heard over the heart.

Subsequently to the last note I repeatedly examined his chest during the many months of his attendance at the Hospital, but beyond some variation in the dryness of the sounds there was no important change in the physical signs. I examined the urine more than once, but the only note I have of it was taken in April, 1869, when it was acid, became slightly turbid on boiling, but cleared on adding a drop of nitric acid.

The left lung remained healthy, and though the patient continued thin and cachectic-looking with a troublesome cough, he held his ground fairly well, and rather improved in general health. At times the expectoration would become very abundant, and occasionally of a pink color, (I thought due to fresh irritation and slight sanguineous discharge from the walls of the old cavity). The most troublesome symptom throughout the case—and one which is common in greater or less degree to all those cases of phthisis in which indurated thick-walled cavities are present—was the paroxysmal cough terminating in vomiting, occurring especially after meals.

No doubt the mechanical conditions of such a cavity, rendering the removal of expectoration very difficult, have much to do with the production of vomiting, and render it a particularly common symptom in these cases; but the reception of food into the stomach has seemed to me to be in many cases so constantly followed by cough ending in vomiting, as to render this mechanical explanation insufficient, and, in 1869, I was led to attribute it to an undue reflex irritability of the pneumo-gastric nerve, and proposed strychnia as the best remedy.[1] I have since, in many cases, found this remedy valuable, but by no means invariably so.

On leaving off attendance at the Hospital in May, 1869, the patient, though not free from cough, continued for a time to improve, but he soon afterwards began again to emaciate, and the vomiting with cough returned. He again attended in January, 1870, resumed the oil and used carbolic acid inhalations, and left March 30th, improved. Since this time I have heard nothing of him.

The above-related case represents very well the main features of fibroid disease of the lung. The indurative disease supervened presumably upon an acute apex (tubercular or catarrhal) pneumonia, and did so with tolerable rapidity, the characteristic symptoms and signs being fully developed within six months of the termination of the acute disease.

The question as to the rapidity with which this disease may advance is one of great interest, and requiring further observation. I cannot but think that, reasoning from the morbid appearances found *post mortem*, we are apt to regard such diseases as older than the clinical history will warrant us in believing; on the other hand, though it is very probable that the fibroid induration of the lung may proceed with great rapidity to such a stage of shrinking as to produce very marked clinical signs, its subsequent progress is very slow and difficult to measure, consisting mainly in the further hardening of an already indurated lung, the gradual widening of the bronchial tubes, and filling up of the loose œdematous areolar tissue between the separated pleural layers by dense fibrous growth. We can readily perceive therefore how the earlier stages of the disease, which are attended with striking alterations in physical signs, may, if the original disease is of considerable extent, be passed through with comparative rapidity, while the later progress is necessarily slow and difficult to estimate.

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[1] Practitioner, vol. i., p. 312:—Dr. Hughes Bennett (Reynolds' System, vol. iii., Phthisis, 1871) regards this as a cause of vomiting in the later stages of phthisis.

There are a few points for consideration in the *diagnosis* of the above case.

That it was not simply a case of contracted cavity is evident from the contraction of the side being general, from the heart being displaced laterally, not specially drawn up towards the right apex, and from the weakness of breath-sound, with dullness at the base, and raising of the liver. The presence of considerable excavation at the apex would be in favor of the disease having commenced there as an apex (phthisical) pneumonia, and not being secondary to basic pleuro-pneumonia or empyema. The same circumstance would also distinguish the case from one of simple cirrhosis of the lung.

The resonance of the opposite lung extending across the median line would emphatically exclude cancer, which the history of the case, and many other signs, particularly the kind of cardiac displacement, would also negative. While in certain cases of mediastinal cancer the heart is fixed in about its normal position, it is, I believe, never displaced towards the side most affected. In regarding the signs of cardiac displacement, however, it is important to avoid taking the point of maximum impulse as necessarily the apex beat. In this case the real displacement of the heart is much less than a first glance would lead us to believe. The absence, while the patient was under observation, of any evidence of complication of other organs would be in favor of the disease being of local origin.

In calculating the *prognosis* in these cases, we have to bear several things in mind—the cachexia of the patient, the size and freedom of communication with external air of any cavities present, and the evidence of disease of other organs, especially of the opposite lung.

The cachexia is occasionally very manifest; without very marked emaciation the anæmia is apt to become great, and the complexion of a straw-tint, reminding one of that seen in the later stages of cases of cancer, or in women suffering from certain uterine diseases. None of these peculiarities were in the present case noticeable in any marked degree.

A cavity of considerable size, and freely communicating with the outer air, is a more hazardous condition for the patient than one which is small, or which we may presume to have become flattened and partially or completely closed. In the former condition the patient is constantly liable, on exposure to cold, etc., to recurrence of irritation and to fresh ulceration of the cavity-wall causing profuse secretion and hectic, as in the present case, or laying bare vessels which may at any time rupture and cause death from hæmoptysis. Out of eight well-marked cases of which I have made *post-mortem* examinations, this has been the cause of death in two.[1]

When the opposite lung is affected, it is most generally by grey tubercle, and it becomes so sooner or later in most cases, unless the patient be cut off by some inter-current disease. We must, however, not give too fatal a prognosis from mere physical signs in such cases, for the course of the tubercle is disposed to be very chronic and indurative, and the signs may again subside and long remain in abeyance. I still occasionally see a patient whose case (Case II.) I brought before the Clinical Society in 1868. He had then distinct evidence of involvement of the apex of the opposite right lung and also a small quantity of albumen in the urine. He has since, however, led a rural life and his health has yearly improved. The right lung has become greatly enlarged, the disease in the left remaining perfectly obsolescent, and the patient is now (1885) in good flesh, and, to all appearance, in robust health.

Sir Andrew Clark has pointed out that albuminoid degeneration of other organs—the liver and kidneys—commonly supervenes in the later stages of this disease. Of the three purest cases of which I have made *post-mortem* examinations, in which there was no obvious grey tubercle in the other lung (though islets of peri-bronchial induration were present in all), in one there was extensive albuminoid degeneration of liver and spleen with granular kidneys; in another of the spleen only, with ulcerated intestines. Albumen in the urine, absent in the present case, affords us the earliest clinical evidence of this degeneration.

Although the prognosis is always, in cases of fibroid phthisis, a precarious one from the circumstances above mentioned, yet the course of the disease may be very long, and may with due precautions in some instances be almost indefinitely extended. The condition of health and *physique* maintained by some patients is remarkably good. In this respect the case related (pp. 213—215) is not so favorable; I have notes, however, of a postman presenting exceedingly well-marked signs of this form of phthisis, who almost entirely lost his cough while attending the hospital, and was able to resume his duties, walking fourteen or fifteen miles a day; and I might mention two or three other patients capable of considerable physical exertion on level ground. Patients who for many years have had one lung dormant or "gone," as they usually describe it, are not uncommonly met with and belong to this category. I have already mentioned one such case which I described at the Clinical Society some years ago, and I occasionally see a lady who has for twenty-five years had her left lung similarly affected. This patient leads a sheltered but useful life and rarely suffers from pulmonary symptoms of any kind; her chief complaint, from time to time, being of failure of heart's action causing chilliness and a disposition to fainting, with occasional attacks of great cardiac oppression which, but for the absence of any severe pain, would be described as angina. There is no evidence in this case of valvular disease or decided dilatation of heart, but the organ is uncovered by the retracted left lung and the sounds are feeble. I have observed in many chronic left-sided cases of phthisis great functional disturbance or rather irritability of heart, doubtless attributable to its being less supported and protected by lung than in health.

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[1] For an account of one of these, vide Clinical Transactions, vol. ii., p. 181, Case 1; the other is referred to in a Table on Hæmoptysis, Pathological Transactions, vol. xxii., p. 58, Case 1, F. W.

The very full consideration of the above examples of fibroid phthisis, and of the general bearings of the disease, renders it unnecessary for me to enter into those variations in the signs of this form of phthisis which depend upon its seat of origin. I will only observe that I believe the commencement of the disease in the central portion of the lung to be of very rare occurrence, much more rare than either at the apex or base. It is true that the most advanced physical signs may often, especially in left-sided cases, be found at the third or fourth ribs, outside the nipple line and in the upper axilla, but this localization of signs is acquired by the lowering of the apex, and shrinking away of the lung from the median line in the course of the contractile disease.

CHAPTER XX.

TUBERCULAR PHTHISIS.

THE following sketch illustrates the phenomena characteristic of *acute tuberculo-pneumonic phthisis—i.e.*, a case of pulmonary tuberculosis, in which the tubercular granulations and groups of granulations are attended with much pneumonia, which latter is the main element destructive to the lung; while the former appears to stamp the disease with its peculiar adynamic characters, its continued fever, and determined progress without check to a fatal termination.

A woman aged 31, had had "inflammation of the lungs" two years ago, but had suffered from more or less cough, with frothy expectoration, for three years. She knew of no family predisposition to phthisis. Four weeks before admission into hospital, however, she expectorated a small quantity, two teaspoonfuls, of blood and the sputa continued to be tinged with blood for five days. She had since suffered from night-sweats, emaciation, cough, and pain in the side and between the shoulders, of which symptoms she complained on admission. The pulse was 112; the tongue furred; catamenia regular. The physical signs on admission were —harshness at the right apex, with sub-crepitant rhonchus; at the left, jerking respiration and prolonged expiration. She lost rapidly in weight, losing 3½ lbs. between October 26th and November 14th.

On November 8th the physical signs were noted as unchanged. On the 21st, she was much worse, had a red, tremulous tongue, a rapid pulse, great breathlessness, and much heat of skin. She could with difficulty stand from the trembling of her limbs and weakness. Sub-crepitant râles were found diffused throughout the right side behind, with some defect in resonance not amounting to dullness. The temperature, taken night and morning from this date, showed a maximum morning height of 103°, average 101.6°; maximum evening temperature 104°, average 102.3°; difference between the average morning and evening temperature .7°. On the frequent occasions when I saw the patient in the middle of the day the skin was uniformly hot, and the pulse very rapid—usually about 120. Meanwhile the pulmonary physical signs advanced, the crepitation became more abundant, and extended through both lungs. There were signs of breaking down at the right apex, though the presence of a cavity could not be ascertained with certainty. On December 19th, there were present "diffused blowing respiration, with sonorous rhonchus and scattered crepitation more abundant at the bases, with some dullness; high temperature, and much dyspnœa." On December 15th, the patient began to be troubled with diarrhœa, which continued more or less to the last. The emaciation or loss of power rapidly increased, the smooth, red tongue became white with aphthous patches, and she gradually sank, having never evinced, however, any delirium or other morbid brain symptoms.

The *continued* fever, the great and early prostration, and the diffused crepitation heard over the lungs, without any defined dullness, rendered the diagnosis of Pulmonary Tuberculosis being the prevailing lesion a tolerably certain one, while the absence of that degree of utter prostration with occasional muttering delirium, and the early presence of decided pulmonary signs, prevented one from regarding the case as one of acute general tuberculosis. There was, however, but little satisfaction to be derived from this reflection, for the prognosis was, so far as present knowledge could decide, inevitably fatal.

Post mortem the lungs were found studded with racemose groups of tubercle surrounded by ill-defined areas of soft catarrhal pneumonia in active process of formation and degenerative softening; the right apex was breaking up into small cavities. There was no miliary tubercle on the pleural surfaces.

The following case of general tuberculosis, the clinical features of which I will sketch, will serve to emphasize the fact that the more purely tubercular affections run their course to a fatal issue with little or no softening, and also to indicate the great difficulty in some cases of making a diagnosis between acute tuberculosis and typhoid fever.

Elizabeth G——, aged 34, a pale, sallow, grave-featured woman, with slight malar flush, was admitted into my ward at Middlesex Hospital in March, 1885, complaining of cough and increasing weakness, with some pain of a pleuritic character in the left side, with which she had been troubled for three months. For a week she had been confined to bed with headache, cough, and some œdema of the feet.

On examination only a few scattered bronchitic râles were discovered in the chest, the splenic dullness was increased, and the abdomen was observed to be full and tympanitic. The pulse was 100, small, feeble, regular; respiration 18; temperature 102·4°. The tongue was very red and raw-looking, and coated in the centre with a thin fur. No spots of a typhoid character were found on the abdomen, but numerous sudamina were observed scattered over the chest and abdomen.

The temperature ranged above 100°, daily reaching 102° and frequently 103°, but was kept more or less modified by antipyrine, administered each time it reached above 102°.

Up to the 20th of April no further chest signs were observed; there was then noted slight dullness and crackle after cough over the right sub-clavicular region, and some superficial crepitant râles at the angle of the right scapula. There was no diarrhœa; the abdomen continued very prominent and tympanitic, and there was some tenderness on pressure, especially over the splenic region.

On April 25th the spleen was felt to extend below the costal cartilages. On May 7th there were signs of a little fluid in the peritoneum, and palpation of the abdomen gave a sense of soft resistance, as though from matting together of intestines by adhesions. Obscure crepitation was heard below the second cartilage on the right side, becoming more manifest and moister at the anterior base. On the left side some crepitations were heard in the mammary region. There were sub-crepitant râles at both posterior bases.

The cough was troublesome, expectoration scanty and viscid. The sputum was carefully examined by Dr. Pasteur (the Medical Registrar) and myself, on several occasions without discovering any bacilli. No diarrhœa was at any time present. The œdema of the legs increased and the patient lapsed into a semi-unconscious state, her temperature moderating somewhat but keeping above the normal.

In this condition she lingered for two or three weeks until death ensued on the 30th June, three months after her admission.

Some of those who observed this case with me from time to time, were doubtful as to whether it was one of enteric fever or tuberculosis. The continued fever, swollen abdomen, enlarged spleen and marked adynamia, were certainly suggestive of enteric fever, nor from experience could it be said that the absence of diarrhœa was sufficient to negative such a diagnosis. The temperature, although maintained at a high level, was of too fluctuating a type to correspond with the assumed period of typhoid. The hectic flush is rarely, if ever, seen and sweatings of sufficient severity to produce sudamina are in my experience never met with in early typhoid. The pulmonary signs were at first quite compatible with either disease. As time went on it was apparent that an adhesive peritonitis with slight effusion was present, and the further development of chest signs rendered the diagnosis secure.

The *post-mortem* examination revealed miliary tuberculosis of lungs, pleuræ, spleen, kidneys and peritoneum, with adhesive peritonitis. There was a small nodule of old disease at the apex of the right lung. None of the tubercles had softened. This patient's sister had ten years previously died of rapid phthisis.

Cases are, on the other hand, sometimes met with in which the catarrhal pulmonary signs in the early weeks of enteric fever mislead to a diagnosis of acute tuberculosis, a mistake which by preventing the strictness of dietary suitable for the latter disease may seriously hazard recovery. Enlargement of the spleen is an important sign in favor of enteric fever, since tubercular disease of the spleen, although present in the above related case, is not usual. Later on in enteric fever the pulmonary signs fade, whilst the enteric phenomena become more marked.

It is to be observed that in this case—not the first in my experience—a careful examination of the expectoration for bacilli on several occasions gave a negative result, and, had importance been attached to the fact, would have misled diagnosis. The ascertained facts respecting the distribution of the bacilli in the pulmonary tubercular lesions would not lead us to attribute any importance to the *absence* of these organisms from the expectoration during the earlier stages of such cases as might most readily be confounded with enteric fever. But in cases of pneumonic type, which are sometimes difficult to diagnosticate from slowly resolving simple pneumonia, the discovery of bacilli will frequently prove of great value in settling the diagnosis.

It is instructive to observe that the *post-mortem* examination revealed some old-standing mischief at the apex of the right lung. In truth, in the infinite majority of cases, if not invariably, tuberculosis is a secondary disease, there having been most commonly a previous abortive attack of phthisis; and when this is not so, there is some caseous remnant of a scrofulous or tubercular lesion elsewhere in the body.

Chronic tubercular phthisis.—In speaking of alveolar catarrh, it was remarked that in some cases it may proceed to catarrhal pneumonia of varying degrees of intensity, in which the pneumonic process gives the prevailing character to the disease. In other cases great thickening of the alveoli, grey induration, in which some individual granules of tubercle may or may not be distinguishable by the unaided eye, is substituted for the more characteristically pneumonic process; we have, in fact, a local pulmonary *tuberculization*, of slower and more insidiously destructive progress than caseous pneumonia, so far as the lung is concerned, but more obstinately and continuously progressive, more prone to be succeeded by early implication of the other lung, supposing both are not from the first implicated, more quickly followed (sometimes even preceded) by disease in other organs, particularly the larynx and intestines; and, in short, though a chronic or sub-acute disease, yet one of more early average termination than the corresponding pneumonic forms of phthisis.

This form of *local tuberculization* spreads through the lung from apex to base, with a well-defined grey advancing margin, immediately beyond which the highly vascular but crepitant lung-tissue presents a striking contrast to it. On examining, however, more minutely with a lens, the alveolar walls are found considerably thickened to some little distance (perhaps half an inch) beyond the defined margin, though the alveolar spaces are not occupied with catarrhal cells—at least, not uniformly so, or to any material extent. A few outlying patches or nodules may sometimes be observed evidently of infective origin. I am not aware of any mere inflammation at all analogous to this in its evasive characters. It most resembles lupus of the cutaneous surface, which, I presume no one would venture to describe or treat as merely inflammatory.[1]

On the other hand, this form of tubercle (as I think it must be considered) differs from miliary tuberculosis pathologically by its primarily attacking one portion of one or both lungs (almost always the apex), and spreading therefrom, not by the dissemination of miliary tubercles far beyond the margin of advance, but by a continuous growth involving the destruction and subsequent excavation of the affected tissue. Clinically, the peculiarly insidious origin and progress of the disease with the gradually increasing *malaise* and anæmia, nocturnal cough, irregular fever, and the physical signs, at first very obscure at one apex, gradually increasing, and developing at the other, are in accordance with its pathology, and distinguish it also from the still more severe miliary form of tubercle. "Chronic tubercular phthisis" seems the best clinical name for this variety, of which the pathological process is, as above stated, best represented by the term pulmonary tuberculization.

It is very difficult to depict in writing a case, with sufficient accuracy to bring out those somewhat minute differences, the accumulation of which build up the distinction between the cases of catarrhal pneumonic phthisis already described, and those of tubercular phthisis, to the pathology of which I have just referred.

It will, then, perhaps be most instructive to enumerate those symptoms and signs the presence of which will warrant us in regarding the case as one of tubercular phthisis—as one, at all events, in which tubercle is the prevailing lesion; for it has been before stated that chronic tubercle is rarely wholly unmixed with other inflammatory products.

So far as temperature is concerned, there is nothing at present known characteristic of chronic tubercle. It is at times elevated, during which periods there are fresh accessions of disease, and the non-febrile intervals are of varying duration. In this respect the disease presents no important difference, so far as I have been able to observe, from the chronic pneumonic forms of phthisis. The physical signs are more characteristic. The obscure signs of alveolar catarrh do not give place to the well-marked dullness and coarse crepitation or crackling of catarrhal pneumonia, but to continued weakness of respiratory murmur, with impaired expansion or actual flattening, while moist sounds may be altogether absent, or one or two dry crackles may be elicited on cough. The percussion-note becomes hardened, and we may be surprised by the appearance (having omitted to examine the patient for a week or two) of some feeble, blowing respiration, of hollow quality, still very dry, which increases in the same obscure way until an unmistakable cavity is present. This formation of a cavity by a process of dry crumbling is very characteristic of the typical form of pulmonary tuberculization.

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[1] Since this comparison was first made in the 1878 edition of my work, lupus has come to be regarded as a truly tubercular affection, perhaps scarcely on adequate grounds.

Huskiness of voice, or actual aphonia, is commonly one of the early symptoms in this variety of consumption, and is then, I think, characteristic of tubercle. The huskiness may clear off, but the voice remains more or less altered permanently in quality. Too hasty a diagnosis must not, however, be made from this symptom, lest a grave prognosis be founded upon a simple laryngeal catarrh. The digestive organs are early affected; the tongue presents a scanty white fur on a very red ground, with prominent red papillæ, an appearance which is very significant of intestinal lesion, still more so if the fur clears off in patches leaving raw-looking glazed surfaces; and the symptoms characteristic of this lesion—alternating diarrhœa and constipation, with colicky pains, especially after food—soon appear.

Patients the subject, of this form of phthisis are usually of slender figures and good features. Among them are those more interesting examples of consumption or decline that novelists prefer to describe. This variety is, however, much more uncommon than the pneumonic form of phthisis.

As to prognosis, these cases admit of considerable temporary relief, and may appear to do well for the first few months. The physical signs progress, however, and I think the duration may be pretty safely reckoned as within two years of the first appearance of definite signs, although I have known exceptional cases to last longer. The intestinal or laryngeal complications cause great distress towards the last, and hasten the fatal termination.

CHAPTER XXI.

ON THE CAVITY STAGE OF PHTHISIS.

THE excavation stage of some cases of phthisis is so prolonged and the symptoms are so decidedly grouped about the cavity, that at some schools it is the custom to name such as cases of "cavitation." Whilst I do not think such a term admissible in any formal sense, however useful it may be as a colloquial expression in clinical teaching, there are yet many points in the diagnosis and treatment of different kinds or conditions of cavities which may form useful matter for consideration in a separate chapter.

We have seen that destruction of lung is the essential anatomical feature of pulmonary phthisis. In the most rapidly fatal cases the destruction takes place simultaneously at many centres, or involves such a large extent of lung as to render hopeless any effort at repair or compensation. In the majority of cases of phthisis that come before us, however, the disease as already pointed out affects principally one apex, the active symptoms attendant upon the pulmonary consolidation and softening after a time subside, the appetite returns and the patient begins to gain strength and flesh. The cough still continues, however, and auscultation reveals the existence of a cavity at the apex concerned; the disease being now usually described as having advanced from the first (consolidation) through the second (softening) to the third (cavity) stage. If these terms were strictly employed in a structural or anatomical sense as regards the lungs only, they would not be objectionable; but in fact they are too often extended in their application to the phthisis of the patient and therefore become fruitful of error and misunderstanding. These so-called stages of the whole disease, phthisis, have reference merely to the effects of that disease upon, perhaps, a fiftieth part, or perhaps, nearly the whole of one or both lungs; they have no meaning as applied to the present or prospective duration of the disease. A man with a big cavity is very frequently better off as regards life and health prospects than one with a "first stage" patch of disease no larger than the area of a shilling. A cavity once formed is so much lung gone, and it is for many reasons much better that the irremediably diseased portion should be cleared out than that it should remain as a centre for fresh irritation, which may break down or infect the system at any time. Our anxiety as regards the immediate prognosis rests upon the condition of the out-lying portions of the affected lung and still more upon the degree of integrity of the opposite lung. Yet the student rarely looks beyond a cavity, upon the discovery of which, in accordance with current phraseology, he classifies the case and decrees the fate of the patient. The physician, too, is often now-a-days beset by anxious enquiries from the relatives and friends of his patient, as to the existence or non-existence of a cavity, upon which they base their hopes and fears, and upon his capacity to discover which, his reputation is registered in their estimation! These terms then, being inaccurate and misleading, should never be used in their general sense.

To resume, however, the special subject of the present chapter. Cavities may be considered under four heads:—1. The recent cavity; 2, the quiescent cavity; the secreting cavity; 4, the active or ulcerous cavity.

Recent cavity.—The recent cavity is the first result of the breaking down of caseous nodules in the lung. Whatever the derivation or constitution of the pulmonary consolidations may be, they, in cases of sufficient intensity, undergo degeneration in few or many centres, and liquefaction of the caseous products ensues. We do not, however, get any physical sign of this liquefaction or of the production of a cavity until communication is effected with a bronchus and some of the softened matter is expelled. From this moment we have cavities existing in the lungs and accessible to the air during respiration.

To yield the auscultation signs which are regarded as necessary for diagnosis, a cavity must have the dimensions of a walnut or larger, and must communicate freely with a bronchus. But on comparing our clinical notes with *post-mortem* observations we shall find the former most commonly inadequate if we have awaited the presence of cavernous breathing and pectoriloquy and such like orthodox signs before admitting the existence of excavation.

The pulmonary consolidations break down into cavities in one of two ways which are not, however, essentially different. Firstly, many minute lobular centres of softening yield to auscultation moist, crackling or humid clicking sounds, which increase in size and abundance as the softening centres extend and coalesce into larger cavities, until finally we get cavernous râle. The respiratory murmur which—bronchial with the first consolidation—had become weakened and more or less masked by the moist sounds, becomes again audible but much altered in quality, now assuming the more or less distinctly cavernous character. As the cavities become fewer and larger by coalescence, the other well-known signs of cavity become apparent—pectoriloquy, splash on cough, *bruit de pôtfèlè*, etc.

Perhaps the large majority of phthisical cavities form and increase in the way I have thus briefly sketched, but in some cases they are formed in a slightly different manner. It not infrequently happens that we fail to get distinct evidence of pulmonary softening for some time after troublesome, but more or less dry cough, and hectic symptoms point very strongly to its presence. There may be dullness, harsh breathing, and some fine spongy crepitation, increased after cough, but none of those distinct clicks characteristic of pulmonary softening. Then the patient will suddenly, in the course of the night perhaps, expectorate a considerable quantity of purulent matter, and we find evidence — cavernous rhonchus, etc., of the existence of a cavity. The explanation of these phenomena is obvious enough: a nodule of consolidation of appreciable dimensions, but rarely exceeding a walnut in size, becomes uniformily caseous, and then softens in its centre and gradually liquefies throughout before communicating with a bronchus, when its fluid constituents are at once expelled, and auscultatory evidence of the existence of a cavity becomes abruptly developed. In the *post-mortem* room we may often cut through such softening nodules in all stages of ripeness for exit; they sometimes undermine and rupture through the pleura, and may well be designated *caseous abscesses.*

Prof. Rindfleisch[1] ingeniously explains the manner in which these softened masses finally communicate with the corresponding bronchus. He observes that there is a general traction upon the branches of the bronchial tree during inspiration, and that as soon as the softening process has sufficiently advanced, a separation is effected between the root of the softened mass and the bronchus which passes into it. Air is immediately drawn into the rent, and the liquid contents of the cavity escape through the bronchial tube. I have satisfied myself that the caseous abscesses above described, do probably discharge themselves by a process of this kind.

With the softening of the pulmonary textures the expectoration ceases to consist of mucus; it is no longer viscid, tenacious, and more or less frothy, but contains opaque specks and purulent streaks, and gradually becoming more purulent each sputum is moulded in its escape through the air-passages, to form a more or less isolated nummular mass. Sometimes, especially as the cavities increase in size, the sputa become more diffluent. As the expectoration increases in abundance, it becomes also easier, and the patient describes his cough as being looser, but soon complains of the amount of expectoration.

At an early period when the centres of excavation are as yet minute, a careful examination of the sputum will discover the presence of elastic tissue in it (Fig. 22). The discovery of elastic tissue has indeed almost exactly the same significance as that of the physical sign of moist crackling, and in all doubtful cases it is well that evidence obtained by the microscope should confirm the stethoscopical signs.[2] Even the most skilled auscultator will often be glad of the help of this instrument.

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[1] Ziemssen's Cyclopædia of Medicine, vol. v.

[2] For method of examining the sputum (see page 44).

Just, however, as I have already more than once remarked, that the physical sign of moist crackling may be produced by the degenerative liquefaction of the products of a *past* inflammatory affection, so the presence of elastic tissue in the sputa does not *necessarily* signify present activity of disease, but simply the removal of its caseous effete products. With completed excavation elastic tissue will no longer be found. Dr. Ewart[1] has pointed out that the elastic elements may become, if not actually destroyed, so much comminuted as to be very difficult to detect in some cases, and this observation I can fully confirm.

A cavity of recognizable dimensions having formed, it may extend indefinitely by new solution of tissue, and by the coalescence into it of smaller cavities, or it may cease to extend. In the latter case it may continue to secrete much purulent fluid for a long time, or it may become quiescent and undergo more or less contraction. The ordinary method of extension and enlargement of cavities by softening down of fresh pulmonary tissue into the original cavity, and by the merging of adjacent smaller excavations into one larger one requires no further comment. It is obvious that all trabeculated cavities have been formed in this way, the trabeculæ being the remnants of the septa which formerly separated the smaller cavities from one another.

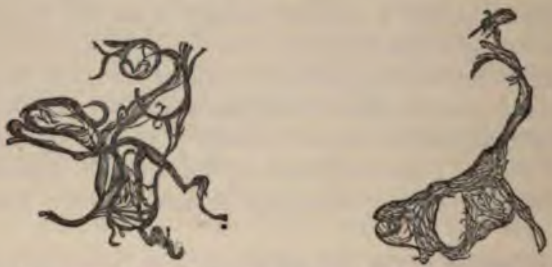

FIG. 22.—Elastic tissue from phthisical sputum. Woodcuts from photographs kindly taken by Mr. F. Fowke. × 170 (about).

There is, however, a theory of Prof. Rindfleisch's by which he explains the enlargement of bronchial and other cavities, which I must notice here although I think there are many and important objections to its acceptance. He regards the obstruction of numerous small bronchi by the pulmonary consolidations as necessitating an increase during inspiration of the air pressure upon the interior of the bronchi in front of the obstruction, and also upon the interior of cavities, thus leading to their dilatation.

Prof. Rindfleisch conceives that the soft walls of recent cavities readily yield before this increased air pressure, and thus enlarge towards the pleural surface condensing the tissue around them. When we consider, however, that the influx of air into the lungs does not take place in any constant quantity, but awaits the aspiration dependent upon the expansion of the thoracic cavity, we see that this theory cannot apply to the enlargement of phthisical cavities nor even to ordinary cases of bronchiectasis. For in both these morbid states, but especially in phthisis, the lung is more or less consolidated or thickened, and having its pleural surfaces adherent, at least over those portions which are excavated, resists expansion more than in health. In vigorous people with healthy lungs the utmost available inspiratory force only exceeds that necessary to expand the lung by from two to three inches of mercury. In phthisis this reserve force is much diminished, the vital capacity of the chest is much lessened, so that we have less air entering the lungs and at less pressure. Hence I think the inspiratory force, effective in producing certain forms of emphysema, has no appreciable action in dilatating pulmonary cavities. I have not observed in phthisis, except in advanced cases with marked dyspnœa, any excessive effort with inspiration; the muscles of inspiration lessen in vigor with the general wasting. During cough, however, the intercostal spaces over a superficial cavity become noticeably bulged, and with the stethoscope we may hear the air forcibly rushing into the cavity, so that doubtless the repeated cough has a tendency to dilate cavities somewhat, but even this is only an auxiliary force in effecting their enlargement.

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[1] Gulstonian Lectures, Lancet, vol. i., 1882, p. 385.

The temperature chart of a case of phthisis with recently formed and extending cavities depicts a markedly hectic type of fever. The temperature mounts up to a considerable height, from 101° to 103° in the course of some hours during the day, the maximum temperature being usually attained at some period between 2 p.m. and 10 p.m. and from this point subsiding to a point below the normal, the sub-normal range occurring usually towards the early hours of the morning. This range of temperature normal to the period of active cavity formation in phthisis has been well illustrated by my colleague Dr. C. T. Williams.[1]

It is sufficiently indicated for clinical purposes by observations taken two or three times a day, provided we are careful to note the period of the day at which the fever is highest and to record a daily observation at that time. We must be further careful to remember that a normal morning temperature means, in these febrile cases, a sub-normal early morning temperature, as this has an important bearing on treatment.

A cavity having formed may at once cicatrize, or may become quiescent and gradually contract, or may continue secreting indefinitely.

In a certain number of cases, cavities, and especially those having their origin in caseous abscesses, *cicatrize*. Laennec describes this as the natural mode in which the pulmonary lesions heal, and seems to regard phthisis as incurable in the first stage. Taking the term cavity in its strict sense, as meaning any loss of pulmonary substance, however small, cicatrization is probably common, nay, we find *post-mortem* evidence of the cicatrization of cavities large enough to come within clinical recognition, but unfortunately they are usually surrounded by other cavities and disease centres which continue to enlarge and render the cicatrization of one amongst them of little avail.

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[1] Medico-Chirurgical Transactions. Vol. lviii., 1875.

I have already (page 202) alluded to a case, however, in which a caseous cavity formed and cicatrized under observation nine years ago in a patient still living, and until recently well.

It more commonly happens that a cavity having attained certain dimensions, becomes *quiescent*, all progress in the pulmonary disease being arrested, its walls become condensed and toughened by the development of fibrous tissue, so as to shut it off from the surrounding lung. The contents of the cavity become less and less abundant, the sounds yielded to auscultation more and more dry, until no moist sounds at all are heard even on deep breathing, but only a few clicks and a characteristic succussion of air are produced on the patient coughing. This kind of cavity at once begins to shrink somewhat in size, its walls becoming denser and thicker by a cicatricial process. The chest-wall flattens and the heart and opposite lung encroach towards the affected side, to make up for the loss of space. If the surrounding lung be tolerably sound it will become expanded around the cavity, so that the latter if only of moderate size and not very superficial, may become altogether obscured. It is very common *post mortem* to see a longitudinal wrinkle or fold upon the surface of the lung bounded by expanded vesicular tissue, and on making a vertical section through such a wrinkle, we cut across a more or less deeply seated cavity, which has evidently undergone contraction. But even superficial cavities may become in this way lost to clinical observation. There are in the Brompton Hospital Museum, some examples of a condition which is well but rudely depicted in Laennec's work.[1] A tough, fibrous band dips into the surface of the lung, and is surrounded by radiating wrinkles between which the pulmonary vesicles are expanded. The band is firmly attached by one end to the summit of a contracted cavity situated half or three quarters of an inch below the surface, and by the other intimately attached to the costal pleura. This band in fact represents an adhesion which originally existed over a superficial cavity but which has become lengthened by the contraction of the cavity and the knuckling inwards of the lung to form the wrinkles described.

That even large cavities may cicatrize and become permanently obliterated is a fact ascertained by *post-mortem* observations, but a close attention to the subject convinces me that this is of very rare occurrence. It is, however, quite common for the physical signs of a cavity which has undergone a certain degree of contraction, to disappear, and be replaced by simply suppressed or very feeble (conducted) breath-sound. This arises, however, not necessarily from the cavity becoming obliterated, but from the bronchus with which it communicates becoming narrowed or occluded by the dense cicatricial growth in the cavity-wall, in which cicatricial growth the sheath of the bronchus partakes. Such a cavity, although it may perchance communicate with a few collateral minor bronchial tubes is practically or completely *closed*, and this is the next best thing to its being obliterated. It diminishes in size and ceases to take any further part in the production of pulmonary symptoms.

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[1] Sir John Forbes' edit., 1827, plate v., fig. 2.

I have not infrequently observed clinically the complete loss of all signs over a cavity of considerable size, and their return after a few days, showing that a temporary closure of the bronchus had taken place probably from a plug of mucus. Probably the periodically abundant and fœtid expectoration found in some cases in which there can be discovered no signs of cavity is due to the cavity still secreting but only communicating obliquely with a bronchus, so that the secretion becomes pent up for some time before it can find an exit.

The *secreting cavity* is usually a cavity of tolerably old date which has ceased to extend and is unaccompanied by active pulmonary disease. It is dense-walled and is lined by a smooth, opaque pyogenic (false) membrane, which can be readily scraped off exposing a highly vascular, dusky, red, subjacent surface. The trabeculæ, which are numerous, present the same vascular surfaces and false membranes. Such cavities may go on indefinitely secreting a diffluent creamy pus, they yield gurgling sounds, with marked amphoric breathing, and dull tubular percussion. Then there is either no fever present or it is trivial, consisting of a slight rise of temperature only at night. The tongue is clean with a tendency to redness and loss of epithelium. Although the appetite usually continues good, the patient slowly loses ground, and acquires the sharp hungry features peculiar to chronic phthisis, with clubbing of the fingers and toes, and a tendency to albuminoid degeneration of organs. Diarrhœa is apt to supervene, and troublesome sickness is sometimes occasioned by the cough. These cases are always of a precarious kind and are not very amenable to treatment.

In favorable cases the secretion dries up and the cavity becomes quiescent.

Unfavorable cases slowly lose ground from albuminoid disease of other organs, or from recurrent diarrhœa. Also, besides the tendency in all cases of phthisis for fresh lung to become involved from a distinct attack, there is in these cases a danger, not commonly recognized, of some of the abundant secretion becoming inhaled during cough into lower parts of the same, or of the opposite lung, and thus setting up fresh centres of disease. The infra-mammary and infra-spinous regions on the opposite side are favorite seats for this secondary disease to appear.

Hæmoptysis is not common from either the quiescent or the secreting cavity, but it sometimes occurs in a dangerous and unexpected manner from the rupture of an ectasia or aneurismal dilatation projecting from the unsupported cavity side of a large pulmonary vessel.

Ulcerous cavity.—The last kind or condition of cavity I wish to refer to, is formed in the usual way, and may have been *quiescent* or merely *secreting* for some time when, from exposure to cold or to septic influences, or from other causes, it assumes a state of active ulcerative extension. I have known such cases to be endemic in a ward which was overcrowded, and to have ceased on a bed being removed. It cannot indeed be too carefully remembered in the treatment of phthisical affections of the lungs that such patients have internal *wounds* or *sores* which, unlike most other internal affections are accessible to the contamination of foul air, and that erysipelatous processes may be readily set up in them, which are apt to be recognized only as *intercurrent pneumonias* or other local inflammations.

Ulcerous cavities are angry-looking, deep, dusky red on their inner surfaces, often studded with hemorrhagic points or ulcerative erosions; they are highly trabeculated and very irregular in shape, but sharply demarcated from the lung tissue by a thin vascular wall. They contain a copious blood-stained purulent secretion which, when expectorated, is mixed with the ropy secretion from the intensely vascular bronchi which communicate with them. The lung tissue surrounding the cavity is injected and œdematous, and at distant parts of the lungs may be found pneumonic centres, which evidently owe their origin to the inhalation of the acrid secretions from the cavity. In such cases there is sharp fever present, with quick pulse, furred tongue, and a tendency to typhoid symptoms. The expectoration is usually mixed with blood, or dark changed clots may be removed from the cavity. Sometimes copious hemorrhage takes place from the erosion of a large vessel.

The prognosis in cases of ulcerous cavity is always grave and hazardous.

CHAPTER XXII.

ON HÆMOPTYSIS.

Hæmoptysis or blood spitting may be defined as the expectoration of blood from or through the lungs or bronchial tubes, and must be distinguished from *false* or *spurious* hæmoptysis, in which the blood is derived from the naso-pharyngeal or buccal mucous membrane.

The causes of hæmoptysis may be thus enumerated:

1. *Hemorrhage from the pulmonary artery or its capillaries.*

(*a*) Rupture or wound of the lung from external violence.

(*b*) Active hyperæmia of the lungs—inflammatory, vicarious, or induced by violent effort or excitement. The active hyperæmia may be primary as regards the lungs; or may supervene or be attendant upon disease—tubercle, cancer, hydatid, etc., already present in them.

(*c*) Mechanical hyperæmia of the lungs, secondary to heart disease, pulmonary overstrain as in whooping cough, embolism of one of the pulmonary branches, or pressure from tumors such as enlarged glands, growths, or aneurism, upon the pulmonary veins.

(*d*) Changes in the blood resulting in capillary hemorrhages—purpuric, scorbutic, toxæmic, (glycohæmia, septicæmia), gouty?

(*e*) Necrotic division of vessels in the course of softening of tubercular or other consolidations in phthisis, tuberculosis, cancer.

(*f*) Aneurismal dilatation or simple erosion of branches of the pulmonary artery exposed in the course of excavation of the lung, or ulceration of the bronchial mucous membrane.

(*g*) Primary atheroma of the pulmonary artery within the lung.

2. *Hemorrhage from the bronchial artery or capillaries.*

(*a*) Capillary hemorrhage from the bronchial membrane of hyperæmic or vicarious origin, or arising from hæmophilia, purpura, scurvy, or toxæmic conditions.

(*b*) Ulceration, erosion, or aneurism, from exposure of a branch of a bronchial artery.

3. *Hemorrhage from the aorta or one of its great branches.*

Aneurism rupturing through the lung or into a bronchus.

The above enumeration[1] will suffice to remind the reader that hæmoptysis is a symptom attendant upon many morbid conditions of heart, lungs, vessels, and blood. For the most part this symptom is included, and sufficiently discussed in the descriptions of the diseases in which it occurs. In some cases of phthisis, however, hæmoptysis from its profuseness, frequent recurrence, and secondary consequences, takes so important a part as to merit separate consideration. Taking out of the above list the causes of hæmopytsis operative in phthisis, they will be found to be—

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[1] This résumé has been adopted from the writer's article on Hæmoptysis in Quain's Dictionary of Medicine.

1. Active or inflammatory hyperæmia.
2. Morbid conditions of small vessels.
3. Erosion or aneurismal dilatation of larger vessels.

Active or inflammatory hyperæmia is generally present in incipient phthisis, and is a prominent feature in the exacerbations of the disease. Hemorrhage is by no means necessarily attendant upon this condition, and does not usually amount to more than coloration or streaking of the sputum.

In the very earliest stage of disease, however, as it affects successive portions of the lung in phthisis, the minute vessels suffer (*a*) softening of their walls by nuclear proliferation; (*b*) more or less narrowing, or even complete closure of their calibre by tubercular growth. It is not impossible that minute aneurismal dilatation may result from this combination of softened vessel wall and increased local blood pressure, after the manner described by Charcot and Bouchard[1] in cases of cerebral hemorrhage, but they have not yet been demonstrated in the lung. The very considerable hæmoptysis in the earlier periods of phthisis, and many of the intercurrent hemorrhages are attributable to one or other of these morbid conditions of vessels, in conjunction with, or independent of, active hyperæmia.

It is very probable that, to some extent at least, we cannot tell how far vaso-motor disturbances within the lungs may contribute to the hyperæmia favorable to the occurrence of hæmoptysis.

As the destructive changes of phthisis advance, the vessels of the affected portions of the lung become softened and torn across, and although, as a rule, their lumen has already become obliterated by previous thrombosis, it occasionally happens that such is not the case, and more or less sharp hemorrhage ensues.

Aneurism of a branch of the pulmonary artery is a common source of fatal, and perhaps frequently also of non-fatal, hæmoptysis.

It is only within recent years that this has been understood, although it was conjectured as possible by Laennec, and more than thirty years ago was demonstrated by Rokitansky.[2]

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[1] Nouvelles Recherches sur la Pathogénie de l'Hémorrhagie Cérébrale.' Archives de Physiologie, 1868. Almost all the cases observed were over 40 years of age, but cases have been recorded at a much younger age. Path. Trans., vol., xxi, xxii.

[2] The first case of pulmonary aneurism was I believe that published by Dr.

These aneurisms are usually situated on a pulmonary vessel which has become exposed as it crosses the wall of a cavity, the aneurism forming a projecting sac from the vessel into the cavity. Loss of support of the vessel on the cavity side, and chronic inflammatory changes in its walls are the causes leading to the formation of the aneurism. The aneurisms vary in size from that of a pea to an unshelled walnut, they may be found at any age at which the suitable conditions for their occurrence are present in the lung. They are most frequently found in chronic quiescent cavities of old date, where the conditions for their formation are best obtained. In acute excavation of the lung, however, they have been met with, although the vessels are more apt to become occluded in such acute processes. A considerable aneurism of the pulmonary artery has been known to occur in a lung in the earliest stage of tubercular disease, affording some reason for the belief that arterial disease sometimes leads primarily to the production of an aneurism which nestles in a pulmonary recess or cavity of its own formation. I had the opportunity of seeing a case of this kind which occurred in the ward of my colleague, Dr. Cayley,[1] in which profuse and recurrent hæmoptysis was the first and only evidence of pulmonary disease for some few weeks. Death occurred from early and acute disseminated tubercle. I can recall a few other instances in which I have found smaller aneurisms nestling in smooth-walled cavities which they exactly fitted, and in which probably the aneurism may have been the primary occurrence. In all these latter cases, however, there was more or less extensive disease of other portions of the lung.

Aneurism of the pulmonary artery may present through the wall of a bronchus,[2] but I am not aware of any cases of aneurism of the bronchial arteries having been reported.

The majority of cases of fatal hæmoptysis have been found to be due to the rupture of a pulmonary aneurism in a cavity. Ulcerative erosion or rupture of large pulmonary vessels within cavities account for the remaining cases.

Bronchial hemorrhage to any serious degree is now generally regarded as of rare occurrence. It may occur in association with the hemorrhagic diathesis, in purpura, in certain malignant fevers, especially variola. Bronchial hemorrhage is sometimes met with in gouty subjects and in alcoholism. Niemeyer and Bürger maintained that hemorrhage from the bronchial tubes was of frequent occurrence in phthisis, and sometimes induced that disease secondarily from the inhalation of blood into the lungs. My own experience leads me to regard both these occurrences as in the highest degree doubtful.

Fearn in the Lancet, Feb. 6, 1841. Attention was attracted to the subject by the cases of Drs. Cotton and Quain, published respectively in the Medical Times and Gazette, Jan. 13, 1866, and Pathological Transactions, vol. xvii.

[1] Case reported in Clin. Soc. Trans., vol. xviii., p. 278.

[2] Case reported by the author, Pathological Transactions, vol. xxii., p. 43.

Syphilitic or tubercular ulceration of the air-tubes leads frequently to slight hæmoptysis, more rarely to copious hemorrhage from erosion of a large bronchial or pulmonary vessel.

Although it is not uncommon for hæmoptysis to be apparently called forth by some unwonted effort or excitement, it is by no means so caused in the majority of cases. Dr. Wilks has noticed the frequency with which hæmoptysis occurs during the night, and perhaps two-thirds of the cases may be said to occur during quietude, and the rest with but rare exceptions only during that degree of physical effort or mental excitement usual in ordinary life. All this points very clearly to the existence of an organic cause at work, the effect of which is but little influenced by surrounding circumstances, the two conditions of weakened vessel and a certain blood pressure being sufficient.

Symptomatology.—Hæmoptysis is too obvious a symptom to require description. The blood expectorated may amount to no more than the slightest streaking or staining of the sputum, or several pints may be brought up.

In cases of decided hæmoptysis the patient is conscious of a gurgling sensation in the bronchial tubes, upon which with a succession of short coughs the blood is expelled in red, more or less frothy, sputa. When more profuse the blood may pour from the mouth in a stream, only partially interrupted by short, gasping coughs. Such hemorrhage may prove at once fatal from its very profuseness, but it is more common in fatal hæmoptysis to observe only a comparatively small amount of blood actually expectorated, the patient at once succumbing to faintness and suffocation from the overwhelming of the air passages with blood. These latter phenomena of sudden and fatal hemorrhage scarcely ever occur except in phthisis of some standing, which has resulted in pulmonary excavation and erosion or aneurism within a cavity.

The blood in hæmoptysis is usually bright red, frothy, coagulating in the receiving vessel in flattened lumps. When very copious it may at the moment of expulsion be dark, venous looking, but this is exceptional. After the occurrence of an hæmoptysis, for some hours or two or three days, the sputa are blood-stained, the color being at first bright and then becoming darker. Sometimes dark, grumous-looking clots are expelled in small quantity from the lung without any previous hæmoptysis, a slight oozing having taken place and the blood being retained some time before expectoration.

In cases of hæmoptysis the shock to the system is very marked, especially in first attacks. The patient is agitated and alarmed, the features pallid, expression anxious, pulse feeble and small, voice partially suppressed, temperature lowered, even to sub-normal. Reaction soon takes place, however, and, especially under the often injudiciously restorative treatment of anxious friends, is ominous of fresh hemorrhage. The pulse becomes large, jerking, and more or less dicrotic, the patient flushed and the conjunctivæ glistening. The sense of weakness and prostration after early hæmoptysis is often prolonged and bears no necessary relationship to the amount of blood lost.

The temperature as already remarked is almost invariably depressed at the first outbreak of hæmoptysis. In cases of parenchymatous hemorrhage—due to hyperæmia and morbid vessels—it usually rises with the symptoms of reaction to above normal within forty-eight hours. In these cases it may be assumed that the depression was a mere fluctuation in the pyrexia attendant upon active disease of the lung.

In cases of quiescent phthisis in which hemorrhage occurs from erosions or aneurisms of a pulmonary vessel in a cavity, the temperature depressed at first, very commonly does not rise at all above the normal.

Again, in apyrexial cases of the latter kind, the temperature may be sometimes observed to rise about the fifth to the seventh day, and the pyrexia may then be attributed to secondary broncho-pneumonic centres from inhaled blood.

Hæmoptysis may of course occur at any period of pyrexial phthisis, when the above-named clinical features attributable to its occurrence may be obscured more or less.

Diagnosis.—The diagnosis of hæmoptysis by a skilled observer present at the time of its occurrence can never be difficult.

1. The blood is distinctly coughed up, is more or less aërated, and either pure or mixed with expectoration.

2. After an hæmoptysis there is generally a staining of the sputa for a few hours or days.

3. Blood expectorated from the lungs is never watery, and non-aërated. (See Spurious Hæmoptysis).

4. In doubtful cases enquiries should be made for epistaxis, and the gums and pharynx carefully examined. (See Spurious Hæmoptysis).

5. Hæmatemesis is frequently associated with a straining, retching cough, but the history of the case, the color of the blood, and the absence of chest signs are sufficient for diagnosis.

6. Only the gentlest possible measures in the way of physical examination are justifiable in cases of hæmopytsis. Percussion must be altogether avoided. The heart's sounds should be listened to, and the breath-sounds over the front of the chest auscultated, without, however, allowing any deep inspirations or other efforts on the part of the patient. By means of a flexible stethoscope the bases of the lungs may be sufficiently explored without disturbing his position.

There will usually be no difficulty in recognizing at one or other apex more or less crepitant or bronchial gurgling râles, significant of the source of bleeding.

The pulse and temperature should be carefully noted, as they furnish the needful indications for treatment.

Prognosis.—In all cases of early or primary hæmoptysis a very hopeful immediate prognosis may be given, since it is the rarest possible occurrence for such attacks to prove fatal. In hæmoptysis occurring in a case of quiescent phthisis in which there are known to be excavations present, the immediate prognosis must be very guarded, but, however profuse the hemorrhage, an absolutely fatal prognosis is never justifiable. The fact of the first outburst not having proved immediately fatal, always suggests the possibility of recovery, for it is certain that aneurismal hæmoptysis is sometimes recovered from.

An ultimate prognosis is a much more responsible matter, and should never be given until the patient has sufficiently rallied to allow of a careful physical exploration, and a survey of all the facts of his case.

Excluding heart diseases, mechanical injury or overstrain, as from whooping cough, acute sthenic pneumonia, and morbid blood conditions, hæmoptysis in the vast majority of instances means existing or threatened phthisis, and is one of its most important positive signs. Numbers of people doubtless make a complete recovery after hæmoptysis, but that recovery is only permanent in those of good family history, and who can and will accept the warning and place themselves under new and more suitable conditions of life. Lightly considered and carelessly treated, hæmoptysis is but the precursor of destructive disease.

CHAPTER XXIII.

ON HÆMORRHAGIC PHTHISIS AND RECURRENT HÆMOPTYSIS.

HEMORRHAGIC PHTHISIS.—Opinions are still divided as to whether hemorrhage from the lungs or bronchial tubes is ever directly causative of phthisis. Niemeyer, Waldenburg, Peacock, Weber, Bäumler, and R. Thompson amongst modern writers, believe with Hippocrates in the reality of a *phthisis ab hæmoptöe*, maintaining that the disease sometimes arises as a direct consequence of blood having been inhaled into the lung from bronchial or pulmonary hemorrhage, and undergoing changes there. Hirtz, Flint, and many others, follow Laennec in denying the efficacy of hæmoptysis in causing phthisis, or rather in holding that the fact of the occurrence of hæmoptysis is a strong presumption of already present tubercle.

Of the possibility of blood having obtained entry to the air-cells and coagulated there, giving rise to broncho-pneumonia and subsequently to phthisical changes, the observations of Niemeyer, Hermann Weber, Bäumler, and Reginald Thompson, have furnished us with clear evidence. I have myself seen on several occasions *post mortem*, examples of inhaled blood forming the nucleus of fresh lobula pneumonia in the grey stage, in cases—it is true of tolerably advanced phthisis—in which death ensued after recent hæmoptysis. And I have no doubt whatever that fresh centres of disease are thus, in those who are already the subjects of phthisis, not infrequently started by hemorrhagic infarction. It is further an undisputed fact that in a certain number of cases, more or less copious hæmoptysis is the very first symptom of the pulmonary disease, preceding, even for a considerable time, all reliable physical signs. Professor Niemeyer asserts that the hemorrhage in these, as in the majority of cases of phthisis in all its stages, proceeds from the bronchial mucous membrane; and that a portion of the blood becomes inhaled into the air-cells of the previously healthy lung, coagulates there, giving rise to irritative lobular pneumonia, the consolidations thus arising, in some cases undergoing decay and leading to destruction of the lung, or becoming cheesy, and giving rise at some subsequent period to tuberculosis of a secondary or infective kind.

Bürger[1] and Waldenburg[2] hold practically the same opinions. I must confess that I have never met with a case which I could distinctly refer to this category, nor are the cases which Niemeyer quotes in support of his view to my mind conclusive. Dr. C. T. Williams[1] and Dr. R. Thompson[2] are also in accord with regard to their experience of the rarity of considerable bronchial hemorrhage. The argument in favor of the origin of phthisis from hemorrhage is considerably weakened by the probability that such hemorrhage, if considerable, is almost invariably of pulmonary arterial source.

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[1] Ueber das Verhältniss der Bronchial- und Lungenblutungen zur Lungenschwindsucht. Tübingen, 1864.

[2] Die Tuberculose, p. 496.

The chief fact upon which those who attribute early hæmoptysis to hemorrhage from the bronchial tubes rely, is the extreme difficulty often experienced by the ablest auscultators in detecting any physical signs (but those, perhaps, of bronchial irritation) a short time after even very copious hæmoptysis. A man comes for examination a day or two after bringing up a large quantity of blood, and absolutely no signs which one could definitely pronounce as indicative of the origin of the hæmoptysis are discoverable. This not infrequently happens. There may be the slightest comparative harshness and feebleness of respiration at the summit of one lung, from which long experience of the subsequent phenomena leads one to judge that the hemorrhage has arisen there, but which without such experience would be considered wholly inadequate to account for the astonishing hemorrhage.

With such very obscure apex signs, after a copious hæmoptysis, there will often be found at one base a considerable amount of scattered crepitation; yet the subsequent development of physical signs takes place at the suspected apex, whilst the basic signs often completely clear up. No doubt the comparative readiness with which, aided by gravitation, the blood can escape from the apex, in a measure accounts for the obscurity of signs there at first, whilst aspiration and gravitation both favor the entry and retention of blood towards the lower portions of the lung. Be this as it may, experience of subsequent development of physical evidences of disease at the apex enables us in such cases to speak decidedly as to their nature, and points very forcibly to the hemorrhage being originally a consequence of preceding disease there, although sometimes a cause of secondary broncho-pneumonic trouble at other parts of the lung.[3]

In the former edition of my work I have at some length described a case of phthisis in a young woman, in which I felt at least justified in affirming that "the *onset* of the disease was *with copious hæmoptysis in a person previously with no apparent chest disease*, and, with the exception of some menstrual irregularity, and the palpitation so commonly associated with this condition, in fair health; and that *a considerable amouut of the disease present is the result of the hæmoptysis;* these two facts being sufficient to mark the case clinically as one of *hemorrhagic phthisis.*"

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[1] Pulmonary Consumption, by C. J. B. and C. T. Williams, 1871, p. 140.

[2] The Causes and Results of Pulmonary Hemorrhage, 1879, p. 11.

[3] Dr. R. Thompson has drawn attention to certain points in the lung which he finds to be the "special habitats" of such hemorrhagic residues and their results; viz., the summit and middle part of the upper lobe, the mid-axillary region between the third and fifth ribs close to the pleura, the anterior inferior border and the middle part of the base, corresponding to the summit of the arch of the diaphragm. Op. cit., p. 50.

I cannot say, however, that subsequent experience would justify me in advocating hemorrhagic phthisis, as thus strictly defined, as an important variety of the disease, since I have been unable to convince myself of its occurrence, save under most exceptional circumstances. The term may be a convenient one in a rougher sense to include cases of phthisis which are marked by profuse and repeated hemorrhage, but thus used it will be found to filch cases from other varieties which have one common symptomatic feature, *viz.*, that of recurrent hæmoptysis.

RECURRENT HÆMOPTYSIS.—The phenomena characteristic of recurrent hæmoptysis are:—

1. The presence of a localized pulmonary lesion presenting no symptoms of activity. 2. Repeated attacks of sudden and severe outbursts of hemorrhage at short intervals, not preceded by any febrile symptoms, and not necessarily attended or followed by any extension of disease.

I may briefly allude to the following cases as illustrative of this form of hæmoptysis.

In May, 1867, there first came under my notice at the Brompton Hospital, a man, aged 27, described as a fitter, who had been ailing for some years, with occasional cough. He complained of pain in the chest and bad cough, but with, he said, no expectoration; he had had streaky hæmoptysis several times. He was doubtful whether he had got thinner; the appetite and digestion were good, the bowels regular, and the pulse slow. His father, an intemperate man, had died of consumption at the age of 44. The patient himself had always been a tolerably steady man of very active habits. He was of sanguine temperament, clear complexion, medium height, and slight though robust build. A striking feature about him, and worthy of note, was his extreme excitability—an almost superfluous energy of character, which led him to do everything with exaggerated effort.

At the date of his first attendance there was present at the left apex some dullness, with a few clicks.

On June 28th he had considerable hæmoptysis which was repeated on August 3rd, and again on the 8th "hæmoptysis one pint this morning," the hemorrhage continuing in smaller quantities until the 17th, after which date it gradually subsided, and having greatly improved in health he ceased attendance at the Hospital at Christmas of the same year.

On the 3rd of August a note was entered of the existence of a small vomica at the left apex, and on the 17th, having regard to the continuance of the hæmoptysis and its repeated occurrence at intervals, together with the absence of any corresponding progress in the pulmonary physical signs, which were still limited to the summit of the left lung, I was first led to suspect the existence of a small aneurism of a pulmonary vessel there.

He continued as he expressed it "well" and at work, until October, 1868, when he returned to the Hospital with slight cough and having expectorated some blood, but not so much as on previous occasions.

At this date there was "dullness anteriorly on the left side to the mamma, with high-pitched bronchial breath-sound, pectoriloquy, and cavernous cough; sounds very dry; some crepitus at angle of left scapula."

He had another slight attack of hæmoptysis in November; and at Christmas was admitted into the Hospital where he remained a month, during which time I had several opportunities of cautioning him against displaying so much energy in doing the most trivial thing, and coughing with such unnecessary violence. He had no appreciable expectoration, and left the Hospital feeling well.

I had never observed any pyrexial symptoms in this patient, and during his short stay in hospital he had a slight attack of hæmoptysis, during which the temperature was observed to be normal.

I did not see him again until August, 1870, when, having remained quite well and at work until the previous Wednesday, he expectorated half an ounce of blood. The physical signs were still limited to the apex, where there was dullness, bronchial respiration, crepitus, and friction (creaking pleura), and a whiffling murmur, systolic, audible in infraclavicular region, continuous from subclavian not from pulmonary arterial region (and no doubt conducted subclavian murmur).

He ceased attendance in October, and continued pretty well until March 4th, 1871, when he again attended with hæmoptysis. Although still spitting blood freely, he, quite against orders, attended personally from Battersea on March 8th, and brought up a considerable quantity of blood in the out-patient room. This attack proved the most prolonged and desperate one he had yet had, and nearly terminated fatally. The hæmoptysis continued with frequent outbursts of half a pint until the 30th, from which date the attacks abated in violence, apparently rather from lack of blood supply than from the efficacy of remedies, which, however, were steadily persisted in until April 13th, when there had been no considerable hæmoptysis for a week.

He again attended personally, though with great difficulty from his extreme weakness, on May 4th. At this date there was noted at the left apex "retraction of lung, dullness, cavernous respiration and rhonchus (slight)." Posteriorly there was "diffused crepitation, with some defective resonance." This was the first occasion on which the lung had appeared to suffer from the effects of the hæmoptysis.

The cough was troublesome, especially in the morning, and on the 11th he was ordered ether and ammonia expectorant in the morning, lest his violent and unaided efforts at expectorating should lead to a re-opening of the broken arterial branch or possible aneurism, which seemed to have been the only conceivable source of such profuse and repeated hemorrhage. It was extraordinary to note the rapidity with which the patient regained flesh, strength, and color, though butcher's meat was only allowed every other day, stimulants were cut off, and abundance of milk alone permitted. He continued to take mineral acids and cod-liver oil. He did not at all approve of the diet, but from previous experience of his rapid blood-making qualities I was convinced that a more generous regimen would have led to a return of the hemorrhage.

On June 29th, having (on the 8th) had one comparatively slight attack of hæmoptysis, the physical signs showed enlargement of the right lung, the margin of which reached across the median line; there was still some irritative bronchitis at the left base indicated by diffused sub-mucous râles.

December, 1871.—Beyond an occasional change in the morning expectoration he has had no more hæmoptysis up to the present time, and has returned almost to his usual health, though the breath is shorter. Since June he has taken no oil; some digitalis was added to his mixture for a few weeks, and the diet has continued restricted, though less so of late.

I have no further note of this man, who did not attend the Hospital again, but I saw him about occasionally and apparently well during the next five years. The case exemplifies well the main features of recurrent hæmoptysis, *viz.*:—1. Repeated copious hemorrhage, obviously arising from disease localized at one portion of the lung. 2. Pulmonary disease, chronic in its course and but little influenced directly by the hemorrhage. 3. The hæmoptysis, *though it may prove directly fatal*, being accompanied by no severe fever or secondary pneumonia, and from it the patient frequently making a speedy recovery.

The pathological condition common, I believe, to all these cases of recurrent hæmoptysis is that of a slowly forming cavity, or one formed by a very localized process of an active character, in the walls of which pulmonary vessels still patent are exposed. I have seen some instances in which an aneurism has presented through the mucous membrane of a bronchial tube, or has occupied the whole cavity of a bronchial dilatation. It will be observed that the case above described did not begin with hæmoptysis; the man had had some dry cough and occasional streaky hæmoptysis for some years previously, and a few days after the first considerable hæmoptysis, a vomica was found at the left apex, where some two months previously there was consolidation and softening.

But the cavity, which yields the blood, need not be of "tubercular" origin—*e.g.*, a soldier was under my care at Brompton for fourteen months, who, in March, 1869, while blowing the clarionet in India, was seized with hæmoptysis to the amount of about a quarter of a pint, which did not quite cease for about a week. A month or six weeks later, after having suffered for four or five days from severe pain and oppression in the right infra-mammary region, he suddenly brought up about a pint of "corruption" and some more blood, and since that time he had had hæmoptysis every few weeks. Whilst he was under my notice, the attacks of hæmoptysis were usually preceded by severe oppressive pain in the right mammary region. The pulmonary disease was mainly at the base or rather the middle of the right lung, there being scattered moist crepitation over the lung, with dullness, most marked at the base. Within the angle of the scapula, and also at the corresponding point in front, opposite the fourth rib, tubular respiration with some large clicks were heard.

This case appeared to have begun with abscess in the lung—whether secondary to pulmonary apoplexy or, possibly, hepatic abscess, it would be difficult to say—which had left behind a deep-seated chronic cavity.

In April, 1872, no copious hæmoptysis for three months, but he had had two or three ounces of current-jelly-like expectoration daily. No evidence of malignant disease. Signs of cavity very obscure.

This patient came to see me again in June, 1876, suffering from a recent bronchial catarrh. He had had no return of the hæmoptysis, and had lost his cough until two months previously. He had gained much flesh, and beyond some dullness and feebleness of respiration at the right base, presented no traces of his former malady.

The danger in cases such as those I have described, is from the abundance of the hemorrhage, which in a great number is the cause of immediate death on the first occasion. It is surprising this should not be so in almost all, and nothing is more striking than the recovery of some patients from what appears to be the most hopelessly profuse hæmoptysis—nature apparently seizing the moment when, from faintness, the blood is at a standstill, to heal the breach by the formation of a coagulum. Hence the importance in treating such cases of withholding all stimulants till the latest moment.

Rokitansky refers to another mode of arrest of the hemorrhage from a large vessel in a cavity—viz., by the cavity becoming blocked by coagulum, which thus compresses the vessel. I have seen an instance *post mortem*, in which the apex of the right lung was converted into a blood-cyst, as large as a lemon, which was quite closed, and which had been produced by hemorrhage into a cavity.

CHAPTER XXIV.

ON FALSE OR SPURIOUS HÆMOPTYSIS.

By true hæmoptysis is meant hemorrhage from the lungs, either from the lung texture proper, or from the lining of the bronchial tubes ramifying through the lungs.

By false or spurious hæmoptysis is meant the spitting of blood which has escaped from some portion of the mucous membrane lining the nasal buccal, or pharyngeal passages. Perhaps the true anatomical line of division between true and false hæmoptysis would be at the glottis, for below this point the mucous membrane assumes the ciliated columnar epithelium characteristic of the bronchial tract, whilst above the epithelium is of the squamous kind. In true hæmoptysis, with the exception of those very rare cases in which the hemorrhage comes from the trachea, the blood escapes from the pulmonary or bronchial vessels; in false hæmoptysis from branches of the carotid trunks. The parts where the blood of false hæmoptysis is derived are the nasal mucous membrane, the pharynx, and the gums and dental alveoli.

1. In cases of decided epistaxis the blood commonly trickles down the back of the throat, and excites cough, by which it is removed in clots and staining the saliva; the source of hemorrhage is obviously, however, the nasal membrane, and no real difficulty in diagnosis ever arises. It is only in cases in which the nasal hemorrhage is but slight, and attended with little or no escape of blood through the anterior nares, that there is any probability of the affection being mistaken for hæmoptysis. This occurrence may happen at night, and the patient wake up spitting blood. The absence of fever and of pulmonary physical signs, and the detection of blood mingled with the nasal mucus when expelled, or of coagula in the nasal passages, will render the diagnosis in these cases also clear.

2. Ulceration of the throat especially when malignant, may lead to copious hemorrhage, and in these cases again, no difficulty is likely to arise in the way of diagnosis.

3. A class of cases is now and again met with which occasions much trouble to the practitioner, and requires much decision in management. These are cases of feigned or hysterical hæmoptysis. Unfortunately, it is too much the custom to regard all cases of spurious hæmoptysis as hysterical, a term for which there is in the majority of instances no possible foundation. For hysterical hæmoptysis is nothing more or less than downright attempt at imposition, the blood being produced either by

sucking the gums, or by pricking or incising them. Dr. Johnson[1] has referred to a case of a young girl sent up to King's College Hospital by a lady interested in her, with an elaborate history of symptoms, including blood-spitting. The character of the expectoration, which consisted of unaërated saliva mixed with fresh blood, was sufficient to indicate its source, and on examining the mouth with a bright light, about twenty fine cuts or scratches were discovered on the mucous membrane covering the hard palate. A sharp reprimand and a short course of shower baths and steel tonics, speedily removed the symptoms.

In some hysterical cases, however, I have known the blood-stained saliva to be highly aërated, probably produced by sucking the gums after injuring them with a needle. The appearance and the physiognomy of the patient is generally sufficient to excite suspicion, and other hysterical symptoms are usually present.

4. A morbid state of the gums frequently arises from want of due attention to the teeth, or from the presence of decayed stumps in the alveoli. On the slighest touch or friction, blood exudes from the mucous membrane, which is swollen and congested, a livid line running along the margin of the gum. A similar condition arises from the effects of certain drugs, especially mercury, and, to a much less degree, lead and iodide of potassium.

In these cases there is fœtor of breath, and an inspection of the gums and teeth at once suggests the probable source of the blood-spitting, which is usually insignificant in amount and unaccompanied by any cough or chest symptoms.

The treatment of these cases falls partly within the province of the dental surgeon, it consists of the employment of astringent tooth-powders, of which one of the best for the purpose consists of finely-powdered kino one part, to three or five of prepared chalk, with or without a little animal charcoal. Any decayed teeth must be removed or stopped. More or less dyspepsia is usually present in these cases, partly arising no doubt from the condition of the gums and teeth, and a stomach cough added to the staining of the sputum may suggest to the patient that he is consumptive.

5. An insufficient supply of vegetable food, a very common dietetic error among all classes of people in towns, leads to a spongy congested state of the mucous membrane of the mouth and fauces, of the same kind as that which, in a more intense degree, is associated with the other lesions characteristic of scurvy. This is one of the most common causes of spurious hæmoptysis. The relaxed condition of the throat, resulting in the secretion of an undue amount of viscid mucus, gives rise to some cough, and the mucus expectorated or rather hawked up from the pharnyx, together with the saliva from the mouth, is from time to time

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[1] Medical Times and Gazette, April 26th, 1862.

tinged with blood. It is often very difficult to distinguish this form of false from true hæmoptysis. And indeed the condition of the mouth is but a sample of that of the mucous membranes generally; and the larger bronchi, if affected with catarrh, are apt to yield a viscid and slightly stained secretion.

I have, moreover, observed in some cases of phthisis a staining of the expectoration which has seemed to me to have arisen in the same way. Anything like considerable hæmoptysis does not, however, arise from this cause, but rather such a staining of the sputa as may suggest fresh congestion or pneumonia.

In the cases under consideration, however, there are no pulmonary signs to be discovered on a very careful examination. The hemorrhage is never in large quantity; it consists of a tinging or streaking of sputum, which is distinctly made up of mucus mixed with saliva, giving rise to a dirty red fluid containing some little streaks or clots of blood. On microscopic examination squamous epithelium cells are seen in abundance, and red blood corpuscles are but thinly scattered over the field. The patient complains of the taste of blood in the mouth, and this is especially disagreeable after sleep. The nutrition is not good, the muscles are flabby and wanting in tone, and the patient feels languid and out of sorts. There is commonly some anæmia present.

A most favorable prognosis may confidently be given in these cases, if we are quite satisfied as to the absence of any pulmonary sign.

In *treatment* the diet must be attended to, an abundance of fresh fruit and vegetables being added. Five or ten grains of citrate of iron should be ordered in fresh lemon juice two or three times a day, and cod-liver oil may often be given with great advantage. Some tannin solution should be used night and morning as a gargle, and to rinse the mouth.

In cases of phthisis in which we suspect this morbid condition of the bronchial mucous membrane to be present, fresh lemon juice, a not unpleasant vehicle for cod-liver oil, is very valuable.

6. In certain cases of anæmia, cases attended with all the other phenomena of that disease, the mucous membrane of the mouth and fauces, although pallid in appearance, exudes a sanguineous fluid, which, mixed with the saliva, causes spurious hæmoptysis. There is in these cases, so far as one can discover, no true hemorrhage from the membrane, no bleeding points can be seen, but in the course of twenty-four hours a considerable amount of blood will transude through the vessels. The transudation is ordinarily very slow, and in the day-time is scarcely noticed, but during the night some accumulation takes place, and on waking the patient expels perhaps an ounce or more of bright red unaërated fluid containing a few coagulated films, giving an appearance closely resembling that of currant jelly and water. Some of the sanguineous fluid often escapes from the mouth upon the pillow during sleep.

The patients suffering from this affection are mostly females: amongst other symptoms of anæmia the menstruation is disordered or suppressed, and commonly, but not always, at the menstrual period the escape of blood from the mouth is considerably increased. Probably from the same cause, *i.e.*, an increased blood-pressure finding relief at the surface of least resistance, any extra exertion is apt to cause an increase in the sanguineous flow. The real pathology of these cases is, however, confessedly obscure; but this is the variety of false hæmoptysis for which we are most often consulted. The patients are breathless, they sometimes have a hard cough, and complain of pain in the left side and considerable prostration, which symptoms, with increasing pallor and blood-stained expectoration, are quite sufficient to persuade them and their friends that they are consumptive.

In such cases again, the greatest pains must be taken to come to an accurate physical diagnosis. The respiratory sounds will usually be found to be weak and partly suppressed from want of muscular power. The percussion is, however, even on the two sides, and the respiration although feeble is vesicular. The character of the cough, both as heard through the stethoscope and otherwise, is usually quite distinct from that of chest disease. Then, there are all the physical signs of anæmia present, venous hum, arterial murmur, etc., and the "pain in the chest" is without much difficulty ascertained to be either infra-mammary neuralgia or gastrodynia. Some patients with this form of spurious hæmoptysis have plenty of color in the cheeks, and are plump rather than emaciated, but they nevertheless present the physical signs of anæmia. The careful observation of a large number of these cases for long periods, enables me to say that it is most rare for them to become phthisical.

The condition calling for *treatment* is the anæmia. More fresh meat must be taken, and if necessary, some pepsine added to aid digestion. In some cases there is considerable disorder of stomach present, which must be first set right before the remedies appropriate to anæmia can be given.

These remedies are the astringent forms of iron. Cold salt-baths or sea-bathing allowed only for a very brief time, one or two minutes, and immediately followed by vigorous frictions, are extremely useful in the convalescent stage. Abundance of fresh air and out-of-door exercise is of course to be insisted upon. The patients and their friends are often much afraid of fresh air, and the cases have usually at the period at which they come under observation been aggravated by confinement in warm and ill-ventilated rooms. It often happens that there are decayed teeth present, setting up irritation in the gums and increasing the disposition to hemorrhage. We must be cautious, however, about advising such to be at once removed, for in these patients the blood is peculiarly aplastic, and hemorrhage difficult to control.

7. General hemorrhage from the whole mucous membrane of the mouth is sometimes seen in hæmophilia. Although doubtless differing chiefly in degree from that described under the preceding heading, there being no perceptible lesion discoverable in the mucous membrane, this hemorrhage is usually so considerable in amount as to prevent any possibility of mistaking its nature. A more or less strongly marked predisposition to hæmophilia is usually also present, and other signs of the disease may be found.

CHAPTER XXV.

LARYNGEAL PHTHISIS—ULCERATION OF THE BOWELS IN PHTHISIS.

LARYNGEAL PHTHISIS.—It sometimes happens in the adult that phthisis is ushered in with laryngeal symptoms, and the variety of the disease thus arising ranks amongst the most fatal and distressing of all.

Strictly speaking the term laryngeal phthisis should be applied only to those cases in which the laryngeal lesion is primary, but in practice it is more loosely employed to include all those cases in which laryngeal symptoms constitute an early and a striking feature. This latter employment of the term is not only more convenient, but is also more in accordance with the general pathology of the disease; for although undoubtedly in many instances the disease of the larynx is the first local manifestation of phthisis, yet in all cases the lungs speedily become involved, so that to attempt more rigidly to restrict the term would imply, what is not the fact, that there is a phthisis which begins and ends with laryngeal disease. Laryngeal ulceration, indeed, most commonly of all arises as a complication in advanced pulmonary phthisis, and in these cases even grave pulmonary lesions are often forgotten in the greater urgency of the throat symptoms, so that they are grouped under the heading of laryngeal, rather than of pulmonary, phthisis. Under this heading then let us, without further regard to logical appropriateness, consider that form of laryngitis which is a part of, or which may complicate, phthisis.

As regards the pathology of the disease authorities differ much, some regarding it as a primary or secondary tuberculosis of the larynx and trachea, in which points of tubercle appear in the submucosa, and, rapidly becoming caseous and softening, produce minute ulcers, which, by their coalescence, form larger ones, whose walls and floors are the seats of fresh tubercular deposits. Other pathologists consider that the lesion commences as a scrofulous catarrh, or inflammation, of the mucous membrane, which speedily involves the submucous glands and determines in some of them caseation softening and ulceration. No doubt the disease occurs in both ways, and the difference between the two forms in the current views of tubercle pathology would not be important. I have myself very rarely met with miliary tuberculosis of the larynx. Laryngeal phthisis is probably in many instances, in part at least, the result of auto-infection through the sputum. Persons who are the subjects of laryngeal phthisis are of highly tubercular predisposition, yet it is remarkable how exceedingly uncommon this form of the disease is in children; nor do laryngeal symptoms commonly occur in children in the course of phthisis.

The morbid appearances found *post-mortem* in the larynx consist of a more or less general inflammation and inflammatory thickening of the mucous membrane of the larynx, and upper part of the trachea, with points or patches of ulceration. The membrane is as a rule hyperæmic, and of a bright red color in the early stages; this color, which fades somewhat with death, is retained in patches of minute vascular injection, especially in the upper trachea. In more chronic cases the membrane becomes more or less dusky or slate colored. In the earliest stage soft and œdematous, the membrane soon becomes thickened and the œdema of a more solid kind, these characters being especially marked in that portion covering the arytenoid cartilages, and in the aryteno-epiglottidean folds; the membrane covering the whole or at least the under surface of the epiglottis is also usually thickened, and the action of these parts is stiffened and impeded by the induration surrounding them. The favorite seat of ulceration is the base of the arytenoid cartilages; a ragged, ulcerous hole is often found in this situation, at the bottom of which the denuded and roughened cartilage may be sometimes felt. The point where the vocal cords meet in front, is also a frequent site for deep ulceration, sometimes extending to the subcutaneous cellular tissue. The mucous membrane above the false cord and over the inner surface of the true cord at its posterior termination is commonly also attacked, and in the more advanced stages all these parts may be involved together. The trachea, especially at its upper portion, and the under surface of the epiglottis, are often the seat of ulceration, spreading from minute points and giving a peculiar worm-eaten look to the surfaces. The cartilage of the epiglottis is not in this form of laryngitis directly attacked, as is so frequently the case in syphilis, but it may lose vitality, and become necrotized in points from deprivation of its mucous membrane. The shallow ulcers below the glottis may also become extensive and deep, causing considerable exposure of cartilages; such an appearance, however, is suggestive of syphilitic complication. The great number of glands dotted over the epiglottis and the trachea explains the peculiar character of the ulceration in both these situations.

SYMPTOMS.—The sufferings of the victim of laryngeal phthisis are great and varied. One of the first symptoms that attracts attention is an alteration in the tone and quality of the voice. It becomes husky and usually deepened in tone. Vocalization is uncertain, the voice sometimes falling into a husky whisper, to come out again, on increased effort being made in speaking, with reverberating deep tone. In some cases, however, even at the earliest stage, the voice is suppressed from an inability to approximate the cords. The patient suffers some oppression of breathing, which may amount to serious dyspnœa, the respirations, and the pulse being both quickened. He looks ill, loses flesh, and suffers from sweats and evening fever. A troublesome teasing cough of a harsh, dry character, attended with but scanty and difficult expectoration, which gives rise to some pain and increases a sense of rawness in the throat, is complained of. Some tenderness is elicited on deep pressure in the upper tracheal region, and a swollen tender gland or two may be felt in the hollow between the margin of the sterno-mastoid and the larynx. Pain on deglutition is a frequent symptom, and it is still more characteristic when discribed as shooting upwards to the ears, causing in them "pricking sensations." As the disease proceeds and the ulcerative destruction of the larynx becomes more extensive, the aphonia becomes complete, and the cough most distressing and paroxysmal. At this period the lung disease has usually proceeded to excavation, and the expectoration is more abundant; at the same time effective cough is almost impossible, in consequence of the patient being no longer able to bring about its essential condition, closure of the glottis. Deglutition is also most difficult and painful from the irritable and stiffened epiglottis failing effectually to guard the laryngeal aperture, and from the pharyngeal muscles compressing the painful and swollen parts.

The fauces and tonsils may be natural looking, they are often pale and drier than natural with their small sub-capillary veins too well-marked. The back of the pharynx is frequently granular looking, sometimes even abraded, with streaks of viscid mucus adherent to the surface. The mucous glands are too prominent. A distinct ulcer having the characteristic raised irregular margin and granular ash-colored surface of the tubercular ulcer may sometimes be seen at the back of the pharynx, or behind one of the tonsils.

An ulcer in rare cases may be present upon the tongue, or on the inside of the cheek, especially about the orifice of the parotid duct on one side. Such cases are by no means necessarily syphilitic, although careful inquiry on this point should always be made. On deeply depressing the back of the tongue with a spatula the epiglottis may, in some cases (those in which deglutition is painful), be seen to be red and inflamed.

But, as I have already observed, the throat, so far as it can be seen by the unaided eye, may be perfectly natural; it is so perhaps in the majority of cases. The pharnyx and palate are sometimes drier and paler than natural, with small ramifying tufts of venules showing out on the anæmic ground. A view of the disease can only be obtained by means of the laryngoscope. Sufficient skill in the use of this instrument to recognize the principal diseased conditions of the larynx, whether these be paralytic or inflammatory, ulcerative or trophic, can be readily gained by a little perseverance and practice. When interference is needed for the removal of growths, or in cases which are attended with peculiar difficulties, more special skill is requisite. With these latter cases I do not venture to deal, but must refer my readers to the admirable special treatises which have been published on the subject.

For ordinary practice, all the permanent apparatus needed is a suitable reflector with throat mirrors of two or three sizes.

Sun-light is the best when it can be obtained. A "Silber" or "Duplex" lamp will yield a sufficient light, but the most suitable of artificial lights (excluding the oxy-hydrogen and other lights which are not easily obtained) is that derived from an argand gas burner, concentrated by a bull's-eye condenser.[1]

The patient should sit upright, well back in a chair, opposite to, and at a little higher level than the observer, so that he is within comfortable reach. The light should be placed near the side of the patient's head, at about the level of his ear, and other lights should be excluded from the room. If sun-light be employed the patient should sit somewhat obliquely with his back to the window. The observer now arranges the mirror upon his forehead so that reflected light will be projected to the back of the patient's throat; he should then direct the patient to open the mouth and put out the tongue, but *not* to hold the breath as most patients try to do during an observation. The tongue should be held by means of a cloth, then gently pulled forward by the left hand, whilst with the right the laryngeal mirror, previously warmed, is introduced. The laryngeal mirror should be introduced (without touching the tongue) with its back to the soft palate, so as to bear back the uvula, and bring the reflecting surface in a vertical line with the glottis whose image it reflects. The condition of the epiglottis and of the vocal cords and their surrounding parts is now seen, and between the cords a view may be obtained of the interior of the trachea.

By directing the patient to say *ah—h—h*,—the vocal cords will be observed in action, and any defect of a paralytic kind affecting the vcoal muscles can be observed; by directing him to breathe deeply the respriatory movements can be noted. If the epiglottis be pendulous, so as partially to hide the cords from view, a gentle traction of the tongue may suffice to elevate it, or by making the patient say *eh—h—h*, the glottis is drawn upwards usually distinctly into view.

There are many little practical difficulties to be overcome which render a satisfactory examination of some patients difficult or impossible with ordinary skill.[2]

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[1] Dr. Felix Semon has designed a laryngoscope in which the handle of the throat mirror carries a small electric illuminator which is fed by means of a small accumulator. Reflectors are thus dispensed with, and a brilliant light is constantly ready, the accumulators only requiring to be recharged every three to five weeks.

[2] I have merely attempted to give an outline of procedure adapted for ordinary cases, and for further details must refer the reader to special treatises on laryngology.

Among the most important precautions and expedients to be adopted are the following:—

1. Before taking any further steps, have the light and the patient perfectly and comfortably arranged, so that a good stream of light can be projected to the back of the pharynx.

2. Take the tongue gently between the thumb and finger, and be very careful to avoid dragging it down upon the teeth.

3. Avoid touching the back of the tongue in introducing the mirror, or retching will be caused.

4. If, as is often the case, the throat be very irritable, desist for a time, and let the patient sip a little ice-water before proceeding again. In cases of great intolerance, one or two applications of cocaine solution, 20 per cent., to the fauces may be employed.

With the aid of the laryngoscope we can thus see at least quite sufficient of the morbid conditions present, to make certain our diagnosis and to prompt our treatment. Acute or chronic catarrh, inflammatory œdema, and thickening and ulceration at one or more points may be observed. The epiglottis often appears thickened and rigid, but we cannot usually see ulcerations upon it, for when present they are as already stated, generally limited to its under surface. Its margin is, however, sometimes roughened or furrowed-looking. Thickening and œdema of the aryteno-epiglottidean folds is most often to be observed, and is considered by Dr. M. Mackenzie to be most characteristic of this disease. In the earlier stages an angry looking spot or two of ulceration, of a yellowish-white color, shining out from a red, tumid, smooth, inflamed ground, may be often seen over the arytenoid cartilages. Ulceration in the more deeply-seated parts may, or may not be seen, but intense injection of the mucous membrane is often visible. In cases of more advanced date, the orifice of the glottis is seen to be distorted by ulceration and thickening, the cords being involved in the havoc so as to render their approximation impossible. In this stage especially, much purulent secretion may collect in the ventricles and about the cords.

The *diagnosis* of tubercular laryngitis rarely presents any real difficulty. The diseases which most simulate it are chronic, alcoholic, or irritative catarrh, syphilitic laryngitis, and hysterical aphonia. The absence of fever, the presence of a definite exciting cause, and the laryngoscopic signs of general catarrh without local thickening or ulceration, are usually sufficient to distinguish the chronic catarrh arising from dust or drink, from laryngeal phthisis. Alcoholic catarrh is moreover always associated with a similar affection of the pharynx. Syphilitic laryngitis is more difficult to distinguish from tubercular, and the two diseases are sometimes combined. Besides the general distinguishing features of syphilis, the local characteristics are larger ulcerations with a less degree of thickening. The ulcers are also more sharply cut than in phthisis.

Extensive destruction of the epiglottis is pathognomonic of syphilis, and much contortion or deformity from cicatricial changes have the same significance.

In functional aphonia the aspect of the patient, the absence of fever, and the soft whispering character of the voice, are in themselves sufficient for diagnosis, showing a defect rather of will than of mechanism. The mobility of the cords is not impaired in this affection, at least in its earlier periods, nor would one expect a true paralysis of the vocal muscles to be rectified by one shock of electricity, as may be observed in these cases.

It sometimes happens, however, that in the early stage of tubercular disease, when there is as yet no discoverable ulceration, the voice is suppressed completely. With the laryngoscope the cords are seen not to come in apposition, in fact they appear to be partially paralysed. The impeded action may be the result of stiffness from the thickening present, but I suspect it is also partly nervous, from the depressing effect of the pulmonary disease reflexly affecting the larynx through the pneumo-gastric. This early suppression of the voice is most likely to be confounded with hysteria; it occurs in nervous people, but the lungs are in all cases more or less involved.

ULCERATION OF THE BOWELS IN PHTHISIS.

ULCERATION of the bowels is a very common complication of phthisis, —indeed it may be said to be one of the attendant lesions of the disease, occurring during some period in the course of a large proportion of cases. Out of one hundred *post-mortem* examinations, of which I have careful notes, ulceration of the intestine was present in forty.[1]

The pathology of the disease is involved in the same difficulty and doubt as that of tubercular laryngitis, opinions being quite as opposed as to whether in its origin it is primarily tubercular or not. In regard to the broader anatomical facts of this lesion, however, there is not much room for difference of view.

The disease commences with inflammatory swelling of the follicles of the small or great intestine, causing them to enlarge and to become choked with their proliferated parenchyma, caseation follows, and the softened products discharge into the intestine, leaving an ulcerous recess behind. The margins and base of this primary ulcer consist "of corpuscular infiltration of the connective tissue, but neither in the corpuscular elements themselves, nor in the mode of their arrangement, can we detect anything specific" (Rindfleisch).[2] Other observers, however, consider this "corpuscular infiltration" as truly tubercular, and others again consider the whole process as being, from the first, of this specific nature. Beyond this point, however, authorities are less at variance. On the peritoneal surface of the intestine, over the site of an ulcer thus established, some flakes of lymph are to be seen, or inflammatory adhesions may be contracted with an adjacent coil of intestine. Beneath the peritoneal surface, and shining through it, the outline of the ulcer can be seen, and in and about its base granulations of tubercle are to be observed. These granulations are connected with the lymphatic vessels, some of which form white lines or streaks over and about the site of the ulcer. The ulceration tends to extend in a transverse direction, beyond the limits of the gland follicle in which it originated. This extension follows the direction of the vessels, and is determined by the formation of tubercles in their sheaths.

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[1] It might have been present in a few more instances, as I have placed a query opposite to some of my negative cases, showing that the intestines were not sufficiently examined.

[2] Pathological Histology, Tuberculosis of Mucous Membrane, vol. i., p. 447, New Sydenham Society's edition.

Whether the ulcers shall be single or in groups depends upon the glands, whether single or agminate, which are attacked. Peyer's patches are favorite seats of ulceration. In them, irregular serpentine ulceration arises by extension and coalescence from several centres, often leaving small tracts or islets of mucous membrane intact. All the gland follicles of a patch are by no means necessarily affected, the ulceration may only involve a certain number of them, extending transversely to the mucous membrane beyond. In the typhoid ulcer, on the contrary, the whole patch is involved simultaneously and uniformly, and the ulceration is limited to the patch. In the cæcum too, the ulceration is often very extensive, the whole of the mucous membrane being eroded for several inches, with the exception of small islets or streaks here and there, which have escaped and stand out prominently, marking the original extension of the ulcerative process from many centres. As already mentioned local peritonitis commonly attends the intestinal lesion, and adhesions are frequently formed. Perforation of the bowel is more common than is generally thought, sometimes leading to the escape of fæcal matter and to general peritonitis, sometimes to the establishment of a fistulous communication between adherent portions of intestine, and again in other cases producing collections of pus localized by surrounding adhesions. These processes are strictly analogous with those which occur in connection with the pleural surface of the lungs in phthisis.

The lower two or three feet of the ileum and the cæcum coli are almost invariably the portions of bowel involved, but the ulceration may extend both beyond and above these points to the rectum and the duodenum. The tendency as a rule is, however, to extension downwards, so far at least as the term extension is applicable to this lesion. In the ileum for instance, the mucous membrane between Peyer's patches is commonly intact. My own observation would lead me to regard the cæcum coli as the favorite seat of this lesion, it is there that tubercular ulceration will be often found when it exists nowhere else in the intestines, and it is there that the lesions, when also present at other parts, are commonly found to be most advanced, and therefore presumably of oldest date.

More or less general hyperæmia of the mucous membrane, very variable in amount at different times, and in different cases, attends the ulcerative process. Considerable general thickening, especially of the large intestines, is sometimes present.

Ulceration of the intestines of the kind above described, is scarcely ever met with in adults and only very rarely in children, save in association with pulmonary disease; it most usually complicates the later stages of the disease; but it may, like laryngitis, occur at a very early period at a time when the pulmonary physical signs—always somewhat masked during diarrhœa—are difficult to detect. Anything that tends to derange the digestion, and the bowels, favors the occurrence of the disease. Klebs[1] and Mosler[2] assert that swallowing the expectoration from phthisical lungs may lead to intestinal lesions. The experiments of Chauveau,[3] who fed heifers upon food containing tubercular matter, and caused ulceration of the bowels and general tuberculosis, favored this view. A great irritability of the gastro-intestinal tract may often be clinically observed, especially in children who do not expectorate properly. Bacilli are present in the intestinal lesions, and have been discovered in the stools. It is *said* that ulcerative disease of the intestines may arise from the imbibition of the milk of tubercular cows. The acid gastric juice is, however, a powerful means of destroying the bacilli, hence the uncertain results of feeding with bacillous foods (*Sée*).

The diarrhœa of phthisis is more prevalent in the late summer and autumn months than in the winter.

The usual symptoms of ulceration of the intestines are *diarrhœa* and *pain in the abdomen.* There is nothing at first which can be noted as peculiar in the character of the diarrhœa; the stools are pale and loose resembling those of ordinary intestinal catarrh, and being dependent doubtless upon the presence of such catarrh. There may be some nausea; the tongue is furred with red tip and edges and prominent papillæ, and the patient complains of thirst. The pain complained of is usually referred to the umbilical region; it is of a colicky nature, but there is no marked tenderness over any spot of the abdomen.

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[1] Quoted in Jones and Sieveking's Pathological Anatomy, 2nd edition, edited by Dr. Payne.

[2] Berliner Klinische Wochenschrift, band x., 1873, p. 509.

[3] Bull. de l'Académie de Méd., t. xxxiii., 1868, p. 1017. The object of Chauveau's experiments was to show the readiness with which the tubercular virus was absorbed by the intestinal canal. They have a very direct bearing upon the question of intestinal lesions originating in the transference of expectoration from the pulmonary to the digestive tract, and they have been confirmed by later observers.

There is marked irritability of the whole mucous tract; the taking of food, and more often warm drinks into the stomach causes the bowels soon after to act. The looseness is, however, at this stage tolerably amenable to treatment, and for a time the motions become natural, or the patient is even constipated. Soon, however, a relapse takes place, and the diarrhœa is more obstinate than before. Now some decided tenderness may be felt on deep palpation, most likely in the right iliac region, the motions become more scanty, some mucus is passed with them, a speck or two of blood may be observed, or a teaspoonful or more may escape. Anything approaching to copious hemorrhage is, however, very rare. The tongue becomes red and patchy from loss of epithelium; it may present short transverse fissures on each side of the median line. The further symptoms vary with the seat of the principal ulceration. If this be limited to the ileum, the diarrhœa may for a long time be held in check by treatment, but the bowels are irritable, the abdomen somewhat prominent in the umbilical region and tender. If the ulceration, as is commonly the case, have its principal seat in the cæcum, the tenderness over that region is more marked, the diarrhœa is very difficult to control, blood and mucus are frequently present in the stools, and the pinched look of the patient and his rapid emaciation are more marked. Ulceration extending further through the colon to the rectum is signified by more distinctly dysenteric symptoms, pain and tenderness over the arch of the colon, tenesmus, more frequent and mucous stools, whilst on the other hand, the gastric symptoms are less marked, and considerable appetite with a fairly clean tongue is often found.

It is easy to note down these symptoms as they occur in case after case of this dreaded complication, but it is much more difficult to fix upon any symptom, if any indeed exist, that is positively characteristic of ulceration. The plain rule is of course, given chest disease, always treat intercurrent diarrhœa as though it were due to commencing ulceration of the intestines.

But in some cases, the diarrhœa precedes, or at the time of observation altogether masks the chest disease. It is remarkable how completely even decided pulmonary disease may be masked by diarrhœa. The cough and expectoration may cease or become trivial, and the dryness of the pulmonary tissue gives an exaggerated "vesicularity" to the respiratory murmur that masks existing defect. The recognition of this fact is of much importance in diagnosis, the alternation in prominence between the chest and abdominal symptoms being very characteristic of intestinal phthisis. So far as my limited experience of dysentery goes, I have also observed that with the appearance of chest symptoms—cough and expectoration—the dysenteric signs markedly abate. It is unnecessary to dwell upon the later stages of this disease; rapid wasting and exhaustion from the constant uncontrollable diarrhœa, a depression of the previously somewhat raised temperature, apthous mouth and lividity of extremities are the closing symptoms.

It is a fact, however, worthy of note that even extensive ulceration of the intestines may exist without any diarrhœa. Dr. Walshe observes: "Not only may pretty extensive ulceration exist in the ileum without pain, either spontaneous or elicited by pressure, but with a confined state of bowels. Again, I have known, in a case running an acute course, marked abdominal pain and tenderness conjoined with obstinate constipation, where, after death, the bowels, in spite too of the frequent use of purgatives, contained abundant solid fæces, and the ileum was extensively tuberculized and ulcerated."[1] I have myself frequently observed constipation to be associated with ulceration of the bowels, most commonly, however, alternating with attacks of more or less severe diarrhœa.

In the *Pathological Transactions* for 1868 I have recorded a case of chronic phthisis with extensive and deep ulceration of the ileum in which constipation was a marked symptom throughout the patient's illness, and I have met with other cases. There is no doubt that the diarrhœa is, to a large extent, dependent upon irritative catarrhal inflammation of the mucous membrane in the neighborhood of the ulcers. In some cases, however, the ulcerations are so deep as extensively to destroy the muscular coat and thus materially to interfere with the peristaltic movements of the intestines. In other cases of a more acute kind, the peritoneum may be much involved, and the muscular coat thus paralyzed. An analogous state of things in both these respects sometimes obtains in typhoid fever. It is not at all uncommon for a case of typhoid fever, well-marked in all other features, to be attended with constipation throughout, and I have known such a case to terminate fatally by perforation.

These latter considerations bring home to us the great importance of a very careful treatment of constipation in phthisis, the stronger purgatives should only be administered with the greatest caution. I have seen perforation occur from the too hasty administration of a couple of the pil. coloc. c. hydrarg. for constipation, which was present together with ulceration. This symptom should be combated with the utmost gentleness.

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[1] Diseases of the lungs, 4th edit., p. 434.

CHAPTER XXVI.

BRONCHI-ECTATIC PHTHISIS.—DUST PHTHISIS—DIABETIC PHTHISIS.

BRONCHI-ECTATIC PHTHISIS.—In some cases of fibroid disease of the lung having its origin in chronic pleurisy, pneumonia or broncho-pneumonia, the bronchial tubes become greatly dilated in the manner already explained (see p. 151). In the later stages of the disease the condensed intermediate lung undergoes softening at certain parts, probably as a consequence of ulceration or atrophy of the protecting mucous surface of the bronchial dilatations. Groups of adjacent bronchial cavities thus intercommunicate to form centres or areas of excavation. This condition with regard to bronchial dilatation is very analogous to the breaking into one another of dilated air-cells in advanced emphysema of the lung.

Coincident with this process of pulmonary destruction there arise symptoms of hectic, emaciation, night-sweats, fluctuating temperature, intercurrent diarrhœa, clubbing of fingers and toes, and sooner or later lardaceous changes in other organs, which, together with the physical signs of anfractuous excavation of the lung in one or more places, constitute the features of a form of phthisis to which the term *bronchi-ectatic phthisis* may be applied. The profuseness and foul odor of the expectoration, and the history of the case will distinguish it from other varieties of phthisis. Elastic elements may with difficulty be found in the sputum but no tubercle bacilli.

Clinically these cases must be ranked as cases of phthisis, and would best be included under the fibroid heading of that disease. Bronchi-ectatic phthisis is always for a long period limited to one lung, but the opposite lung is rarely sound, its actual condition varying according to the mode in which the disease has arisen. In cases in which the bronchi-ectasis is secondary to a pneumonia or pleurisy, the disease may be limited to the lung first affected, but even in these cases the enlargement of the second lung is in many instances of the nature of emphysematous dilatation rather than compensatory hypertrophy, and in the later stages of the disease it becomes affected with secondary broncho-pneumonia from putrid absorption or inhalation of acrid secretions, or it may be involved in secondary tuberculosis, and this secondary disease may proceed to the formation of cavities. In cases in which the bronchi-ectasis is secondary to broncho-pneumonia, the second lung almost always presents some thickening of its interstitial tissue, fine, fibrous processes branching through the lung from the bronchial sheaths, and more or less bronchial dilatation. But the supervention of more active inflammatory changes in the cirrhosed lung, necessitates compensatory dilatation of that less affected, and the emphysema thus brought about masks, and in a measure no doubt checks, the fibrosis there present. That the enlarged lung is not attended with increased function of a truly compensatory nature is, however, manifest from the breathless condition of these patients.

When once the lung has become broken into, and the sypmtoms described have set in, the cases are but little amenable to ordinary methods of treatment. It is possible that with the increased attention now being given to pulmonary surgery more may be done for these cases than has hitherto been accomplished in the way of drainage, and the promotion of cicatricial obliteration of the chief cavity centres. It is at the basic portions of the lung that such excavations especially require interference, for to their very inadequate natural drainage is chiefly attributable, the fœtor and acridity of sputa and the hectic phenomena. When situated at the apex, and unless, as sometimes happens, the chief bronchial channel is at its entry to the affected lobe pinched by cicatricial contraction, the natural drainage is probably better than any that could be effected artificially. It may still be a question, however, whether a curative process may not be induced by the introduction of a tube from without.

There is a certain liability to cerebral (embolic) abscess in these cases, even without operative interference and any slight additional danger from this cause must, with other dangers connected with an operation, be carefully weighed against the deplorable plight to which some of these patients are reduced, as their disease advances.

DUST PHTHISIS.—The more or less constant exposure to the dusty atmosphere in certain industries results in serious damage to the broncho-pulmonary system.

1. I have already described (page 107) a case of dust bronchitis, asthma and emphysema, which illustrates the disease most common amongst those engaged in dusty employments. In the first generation of persons thus employed, when the family history is free from chest diseases, a certain degree of tolerance is often established which enables them to continue at work, but they always suffer from chronic bronchitis and emphysema, with increased bronchitis during the winter months, and rarely attain old age.[1]

2. It very commonly happens, especially in those predisposed to phthisis, for an alveolitis to be set up, either through direct mechanical irritation, or perhaps more frequently through susceptibility arising from

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[1] In the public health reports for 1860-61, and in the Pathological Transactions, vol. xx., 1869, will be found original investigations into the etiology and pathology of pulmonary diseases arising from the inhalation of dust, and references to the literature of the subject by Dr. H. Greenhow, F.R.S.

constant broncho-pulmonary irritation. A catarrhal pneumonia is thus produced, usually of chronic progress and very fibroid type, resulting in dense fibroid induration of the affected lung, with slow necrosis and excessive thickening of the pleura. A large amount of pigmentation of the lung, producing a slate-colored mottling of the consolidation, is common to all these chronic cases, but in those cases in which the particles inhaled are of carbonaceous nature, as in colliers and miners generally, in addition to natural pigmentation, the tissues of the lung are infiltrated with carbon inhaled from without. It is stated that some of the carbon thus deposited has its source in defective elimination of carbonic acid, in consequence of the highly impure atmosphere in which some miners spend much of their time. In some cases the minute bronchioles become choked with carbonaceous matter.

Cases of dust phthisis are most generally one-sided, and present all the features of fibroid phthisis. They are of very chronic course, attended with but little pyrexia. The cough is paroxysmal, and the expectoration difficult, consisting at first more or less of pigmented bronchial mucus, becoming later purulent. In highly carbonaceous cases the expectoration, especially during the softening stage, is black, and yields an abundant deposit of insoluble carbon. Observations have not yet been made as to the presence of bacilli, but the conditions present are not favorable to these organisms.

3. Phthisis of the ordinary catarrhal form is frequently developed in those young operatives in dusty factories, who have a family predisposition to the disease, which the conditions of dusty atmosphere and confinement in ill-ventilated rooms strongly tend to develope into disease. These cases do not present any unusual features.

DIABETIC PHTHISIS.—Destructive disease of the lungs occurs as a frequent and fatal complication in diabetes. The statistics brought forward by Drs. Finlay, Stephen Mackenzie, F. Taylor and Dawson Williams, at a recent discussion on diabetes at the Pathological Society,[1] show that about thirty per cent. of cases of diabetes die from phthisis, most commonly in the second to the fourth year of the disease, and that in a still larger proportion, nearly one-half, pulmonary lesions are present.

That the occurrence of phthisis has some specific relationship to the diabetic state, has been argued from the fact that as the pulmonary destruction advances the sugar in the urine diminishes, and may even disappear, and in those cases in which no pulmonary disease devélopes, death is more apt to ensue from coma (Mackenzie).[2] Dr. Dickinson[3] who with Wilks and Pavy, regards the pulmonary lesions in diabetes as of non-tubercular inflammatory nature, considers their origin as probably attributable to some disturbed innervation of the vaso-motor system. This is of course at present a matter of speculation only; one is reminded, however, of the possibly analogous subsidence of the paroxysms in asthma with the supervention of phthisis.

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[1] Trans, Path. Soc., vol. xxxiv., 1883.

[2] Loc. cit., p. 357.

[3] Diabetes, by W. H. Dickinson, M. D., 1885.

Notwithstanding the frequency with which phthisis occurs as a complication in diabetes, the occurrence of diabetes in the course of phthisis is exceedingly uncommon.[1] It is therefore apparent that whilst diabetes is a potent cause of phthisis, on the other hand phthisis has no tendency to produce diabetes, so that the causative relationship between the two is not reciprocal any more than is the causal connection between syphilis and many other morbid or depressing conditions and phthisis.[2]

In its clinical features as well as in its essential pathology, diabetic phthisis comes within the catarrhal pneumonic group, and there is to be found no real difference between the "chronic caseous broncho-pneumonia" (Dreschfeld)[3] of diabetes, and catarrhal phthisis as already described. The formation of cavities in diabetic phthisis often takes place with great rapidity, as might be expected from the aplastic and highly necrotic tendencies which are so strongly marked in all the inflammatory lesions of diabetes. Dr. Dreschfeld, who has given much attention to the subject of pulmonary complications in diabetes, characterizes the caseous broncho-pneumonic lesions as tubercular, and has found in them, *post mortem*, tubercle bacilli. He also refers to Immermann and Leyden, and Frerichs, as having found bacilli in the sputum. Dr. Dreschfeld has, however, observed two cases of chronic croupous pneumonia, resulting in excavation of the upper lobe in diabetes which presented the clinical signs of phthisis, but in which no bacilli were found either in the expectoration or in the lesions *post mortem*. This non-tuberculous form of phthisis is of much more rare occurrence than the other.

Phthisis is more apt to occur and runs a more rapid course in the diabetes of young people. Under all circumstances, however, it is a complication of very grave augury.

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[1] Bertail, Étude sur la Phthisie Diabétique, 1873, p. 11. Also Bouchardat, Etiologie de la tuberculose pulmonaire, quoted by Bertail.

[2] See also the remarks by the author in the discussion on diabetes, op. cit., p. 394.

[3] On the Pathology of the Lung complications in Diabetes, by J. Dreschfeld, M.D., Medical Chronicle, vol. i., p. 5, 1884.

CHAPTER XXVII.

SYPHILITIC DISEASE OF THE LUNG—SYPHILITIC PHTHISIS.

THE respiratory organs may become affected in the secondary or tertiary periods of syphilis.

The bronchial mucous membrane is sometimes involved in the eruptions of secondary syphilis, much more rarely at this period in ulceration. It is true that the positive evidence of macular syphilis of the bronchial tubes is incomplete, and not confirmed by *post-mortem* observation, but very decided symptoms of bronchial catarrh are so frequently met with in association with the secondary cutaneous rash of syphilis and the corresponding throat affections, that it is impossible to escape the conviction that a specific catarrh of the respiratory tract is present in such cases. These catarrhal symptoms and the mottled cutaneous eruptions, together with a certain degree of pyrexia, may sometimes be readily mistaken for measles.

I have not observed asthma in association with the bronchial eruption in syphilis. Syphilitic ulceration of the bronchial tubes is of rare occurrence during the secondary period of the disease, and is only occasionally to be met with in association with other tertiary lesions. When a main bronchus or the bifurcation of the trachea is the seat of a syphilitic ulcer, paroxysmal cough and dyspnœa, with scanty expectoration occasionally streaked with blood, may be expected, and the diagnosis would be helped by the observation of other phenomena of syphilis. The ulcer may perforate the trachea or bronchus and produce mediastinal or pulmonary abscess; a branch of the bronchial artery may be opened up leading to severe or fatal hemorrhage. The tendency of the syphilitic ulcer is, after deep erosion of the tissues, to cicatrize, causing great contraction and deformity of the tubular structures affected. I have already alluded to narrowing of the bronchi as one of the dreaded consequences of syphilis (see page 148). Syphilitic ulceration of the smaller tubes is of occasional occurrence and may extend into the lung, producing peri-bronchial consolidations from pneumonia.

The clinical recognition of bronchial syphilis rests (1) upon the presence of symptoms of broncho-pulmonary disease which are not in the order of those characteristic of simple bronchitis or phthisis; (2) upon the manifestation of the syphilitic cachexia in other directions and in the history of the patient; (3) the rapid amendment, provided the disease be not too far advanced, that ensues upon anti-syphilitic treatment.

The lung proper is affected with syphilis:—(1) as already pointed out by extension from ulceration of the bronchi; (2) by gummatous nodules (syphiloma); (3) by diffused pulmonary fibrosis extending from many centres of gummatous induration; (4) by gummata of the pleura extending into the lung along the inter-lobular tracts.

The lung may be affected either in *congenital* or *acquired* syphilis. In the *congenital* form of syphilis, gummatous nodules are sometimes met with in the lungs. In other cases the lesion takes a more diffused form, occupying a considerable area of lung, usually in its central portion or at the base. Out-lying nodules may also be found. The diffused consolidation which at first sight resembles pneumonia, consists of tracts of granulation tissue with interlacing bands of fibres and alveolar and bronchial thickenings. The alveolar epithelium takes but small part in the consolidation, the alveoli being collapsed by the interstitial fibro-nuclear growth upon which fatty degeneration ensues, sometimes producing centres of softening. The syphilitic growth is at first permeated by vessels which, however, in the later stages become extensively obliterated. The penetration of syphilomatous growths by vessels is a mark distinguishing them from tubercular lesions.

The lung affected by this diffused syphiloma is heavy and solid, of greyish-white color and smooth section, very different from the granular friable surface presented by croupous pneumonia. Adults are very rarely affected with this form of extensive lung consolidation from syphilis.

Congenital syphilitic lung disease in infants is a matter more frequently of *post mortem* than clinical observation, the cases in which it occurs being most generally fatal from other visceral lesions and from general cachexia; hence it is of small clinical importance. No doubt congenital syphilis has something to do with catarrhal and even phthisical lesions which develop in later childhood and adult life, but the measure of its influence in such affections cannot be definitely traced.

Acquired syphilis of the lung makes its appearance generally in from four to six years or more after the primary infection. I have known twelve years to elapse before the manifestation of pulmonary lesions.

The *symptoms* of pulmonary syphilis are often very obscure and may readily be confounded with those of mere ordinary inflammatory conditions. Gummatous nodules have been found in those who during life presented no evidence of pulmonary disease. The symptoms generally present are those of chronic indurative disease of the lung or pleura, *viz.*, paroxysmal cough with difficult expectoration, of obstinate continuance uninfluenced by ordinary remedies, which is yet associated with but little emaciation and no pyrexial phenomena. Pleuritic pains are often present. Hæmoptysis is rare, and when it occurs is highly suggestive of phthisical complications. The presence of such symptoms in a person bearing the marks, or giving a history, of syphilis would be very significant.

It not infrequently happens, however, that some peculiarity or incongruousness in the symptoms or physical signs leads to the discovery after close inquiry of a syphilitic taint, the surface marks of which are very obscure, and the history of which is at first denied either intentionally or through ignorance.

The most characteristic *physical signs* are those of localized pulmonary induration, *viz.*, flattening, dullness or a sense of *hardness* on percussion, with enfeebled breath-sound of blowing quality with few or no moist sounds. Some bronchial clicks, or, more commonly, a superficial crackle of pleuritic or sub-pleural source may be heard. Such signs, when presented at some unusual situation, as about the mammary or infra-mammary or infra-spinous region, are very characteristic. It is certain, however, that in a large number of cases the physical signs are not localized in any unusual spot, but present themselves at one or other apex. The diagnosis under these circumstances from chronic tuberculization is very difficult. The evidence, however, of considerable one-sided indurative disease of the lung, of chronic course, and not traceable to any preceding acute attack, in a person who has not been exposed to any dusty employment should lead to a suspicion of syphilis, and, having arrived thus far, a careful inquiry into the history and examination for surface marks will usually clear up the case. No bacilli are to be found in pure syphilitic disease of the lung.

Syphilitic gummata sometimes soften and produce cavities, which are small, however, and often too deeply seated for recognition. In such cases hæmoptysis may occur from the formation of pulmonary aneurism. The treatment of pulmonary syphilis consists in the steady administration of mercury in small doses, in association with or followed by a course of iodide of potassium. The mercury may be given in the form of the perchloride or in many cases better by inunction. A few seasonal courses at Aix-la-Chapelle may be advised. In all cases, however, the general health of the patient must be well looked to and maintained.

Syphilitic phthisis is rather clinically than pathologically a variety of consumption. There can be little doubt that cases occur, perhaps with greater frequency than is suspected, in which syphilis of the lung remains untreated, being from the first mistaken for tubercular disease; and destructive changes ensuing, the phenomena of softening and excavation present themselves, and are associated with the hectic phenomena and wasting characteristic of phthisis.

The cases of syphilitic phthisis, however, which are most frequently met with, are really hybrid examples of phthisis engrafted upon a more or less strongly marked syphilitic cachexia, in which the phenomena of syphilis are manifested with, and in a measure modify and control, those of phthisis. Cases of syphilitic phthisis, as thus defined, present the following peculiarities. Hæmoptysis is in a large proportion of cases an early and an urgent symptom, the hemorrhage being severe and recurrent. The cases usually present the caseous pneumonic type, being one-sided, with rapidily advancing consolidation and softening, with subsequent arrest and fibroid changes. The evidences of lardaceous change present themselves at a relatively early period, and with greater proportional frequency in the syphilitic than in the other forms of phthisis. The physical signs are somewhat peculiar, more especially so in those cases in which the syphilitic cachexia is marked, much less so in those cases in which the syphilis has merely served to weaken resistance in one with strong hereditary predisposition to phthisis.

In fairly well-marked cases of syphilitic phthisis there is in the earlier stages over a widely extended area a fine superficial crepitant râle suggesting a sub-pleural origin, and out of proportion to the signs of positive softening present. Sometimes this superficial crepitant râle will extend in a patchy manner over almost the whole side, being associated with but little or no percussion dullness. In the later stages again, the degree of fibroid thickening of lung and pleura is excessive. The patient may present other marks of syphilis, *e.g.*, cicatrices upon the tongue or throat, nodes, cutaneous scars, or pigmentation. His build and conformation of chest are often not those of ordinary phthisis, and the complexion has the earthy, lustreless pallor of the syphilitic, rather than the hectic aspect of the phthisical cachexia. In other cases again, the patient presents the aspect typical of advanced consumption, when the pulmonary lesions discoverable are but slight. In these latter cases perhaps excessive activity in anti-syphilitic treatment may have prostrated a patient of tubercular constitution.

Some stress has been laid, especially by army surgeons, upon the presence of enlarged posterior cervical glands as an evidence of syphilis. In looking for this sign as an aid to diagnosis, I have, however, been too frequently disappointed to enable me to assign to it much value. In cases in which the larynx is involved a laryngeal examination will disclose the more cleanly cut and widely destructive ulceration of syphilis, or in later and less active stages the evidence of scarring and deforming cicatricial changes.

CHAPTER XXVIII.

ON OTHER IMPORTANT COMPLICATIONS OF PHTHISIS—TUBERCULAR MENINGITIS—LARDACEOUS DEGENERATION OF ORGANS—ALBUMINURIA—FISTULA.

TUBERCULAR MENINGITIS.—This fatal malady most commonly occurs as a complication of phthisis, as part of a secondary general tuberculosis. When the meninges of the brain are importantly involved in this outbreak of tubercle, the special symptoms that arise are so grave as to set aside from view all other conditions present.

There is no more striking feature about tubercular meningitis when it complicates phthisis than the uncertainty and insidiousness of its supervention. It is happily a somewhat rare disease, yet there is no case of phthisis, and no stage of any case in which the conditions for its possible occurrence are not present. A child may have had an apex catarrhal pneumonia resulting in some induration and flattening with complete subsidence of all symptoms; she daily improves, and leaving off all treatment, is regarded by her parents as well; but the doctor says that her chest is still delicate. She suddenly developes brain symptoms and is carried off in two or three weeks. This is a common history.

I will briefly relate one or two cases which may be regarded as typical of their kind. George B——, aged twelve, a school-boy, admitted into the Brompton Hospital under my care in August 26th, 1875. He was a thin, pale, somewhat underfed, neglected-looking child, and his present ailment dated from an attack of measles four years before, which was followed by a cough, the expectoration at that time being occasionally streaked with blood. The cough had remained more or less since. A month before admission to the Hospital he had kept his bed for a week with pain in the right chest, and since that time had been troubled with night sweats and loss of flesh.

At the time of admission the boy was but little troubled with cough; his tongue was slightly coated and bowels confined, but he had a fairly good appetite. The respiratory murmur was weakened over the whole chest and accompanied by slight occasional sibilus. The left base did not expand with inspiration, and there was defective resonance on this side below the nipple level in front, and in the axilla, with more decided dullness below the same level posteriorly. On the right side there was dullness at the extreme posterior base for three fingers' breadth.

The patient was considered to be suffering from slight bronchitis with the remains of broncho-pneumonia affecting the left side principally.

There was nothing in the case to attract further notice until the 18th September, when vomiting after food was complained of. On the next day the vomiting continued, and there was some diarrhœa. On the 20th, pulse 84, respirations 20, temperature 99° at 8 p.m. Had vomited six times. 21st. No vomiting since last night, *urine* yielded on boiling a heavy cloud of *phosphates*. The vomiting persisted through the 22nd, 23rd, and 24th, the tongue being white and coated, with prominent papillæ. *No headache or pain.* 25th. Vomiting continues. Face flushed, skin hot, perspiring.

On morning of the 27th the patient became *unconscious*, and could scarcely be roused to put out his tongue, pulse 84. At 4 p.m. my note was as follows. "Pulse 76, *irregular*, lips dry, tongue furred. Expression of face *drowsy, suspicious.* Will not answer questions. Tries to put out tongue when sharply told to do so, but fails. Is slightly delirious. Respirations 20 in the minute, temp. 100·8°. There is slight occasional *twitching of the left arm* and pectoral muscle. When aroused drinks oatmeal water with greediness. Evidently tries to answer questions put to him." 9 p.m.—Twitching of both arms and legs, picking at bedclothes, grasping at nothing. Has not vomited after taking cream and brandy, pulse 80, temp. 100°.

Sept. 28th, temp. 100.2°, pulse 80, more restless. Fingers continually working, scratching or pulling at teeth. *Muscles of back rigid.* 4 p.m. Movements of arms and legs more violent. Rigidity back more marked; temp. 102·2°, bowels open from medicine, motions loose. 29th, temp. 101.2°, pulse 80, weaker. Slight external strabismus of left eye. 30th, 10 a.m., temp. 102.4.° 7 p.m. 103,° pulse 120. Eyes roll slowly from side to side, pupils dilated. Oct. 1st, temp. 101.6° a.m. Oct. 2nd, temp. 101.6° a.m. Increasing emaciation. Some resistance to extension of arms. Conjunctivæ more sensitive, pulse 130, *regular*. Patient lingered for a week longer in much the same state and then sank.

Post mortem.—The cerebral convolutions were found to be flattened, the pia-mater over them congested. The ventricles were distended with clear fluid, the ventricular walls being soft, almost diffluent, the choroid plexus not congested.

At the base of the brain the pia-mater was congested over the anterior and middle lobes, and presented innumerable miliary granulations. Over the central space between the middle lobes the membrane was thickened, opaque, and contracted. The left middle and anterior lobes were intimately adherent at the fissure of Sylvius by thickened, opaque, yellow lymph; the veins in this fissure were blocked, and immediately under the pia-mater the cerebral substance was softened down into a cavity. On the opposite side in the same position, the brain was much softened.

In this case during the first days of the attack no headache was complained of, nor did the boy at any time complain much of this symptom. Vomiting of food was the first, and for the first week, up to the period of commencing unconsciousness, was the only important symptom that might direct attention to the head. But in a boy it would first, at all events, direct attention to the stomach, and being associated with furred tongue and disordered bowels, it was attributed to derangement of this organ. It, however, obstinately resisted all treatment. The first circumstance which led me in this case to attribute the vomiting to its right cause was the observation by my clinical assistant, that the urine which he had examined for albumen yielded on boiling a heavy cloud of phosphates. This was on the 4th day of the vomiting. Twitchings and irregular movements of the limbs with increasing unconsciousness supervened on the 9th day, with some rigidity of muscles, especially those of the back of the neck. The temperature which was not elevated at first was distinctly so from the 9th and 10th days. Squinting and latterly blindness were present in this as in most other cases of tubercular meningitis.

The following are the main features of another case which was under my observation in the Brompton Hospital from Dec. 12th, 1876, and previously as an out-patient at Charing Cross.

A man, aged 33, predisposed to phthisis, had a cough with expectoration in February, 1876, and in April began to suffer from fistula. He improved in health for a time, but the fistula continued to discharge freely, and in the late autumn the cough and expectoration were more severe, the hectic symptoms more marked, and physical examination showed that he had sub-acute phthisis, affecting principally the apex of the left lung. At Christmas a sub-divided cavity was found to exist at this situation, and the opposite lung, although enlarged, was partially consolidated at its summit. The pulmonary symptoms were now, however, quieter, and the patient referred his slowly increasing debility to the discharge from the fistula. On the 10th of January, weak iodine injections were used with the effect of somewhat lessening the discharge. Before the employment of these injections, however, the temperature had latterly been somewhat raised and the cough more troublesome. On this 10th of January the patient first complained during the night of slight headache and confusion of vision, which was attributed to a small dose, $\frac{1}{100}$ of a grain, of hyoscyamine given in a linctus at bed-time to relieve the cough, and which had caused some dryness of throat. But in the morning the pupils were not dilated. The temperature was raised, and the pulse 100; cough lessened, but expectoration tinged with blood. The hyoscyamine was repeated at night, and gave much relief to the cough, but the headache returned with severity, and in the morning (12th) the patient vomited some bilious matter. Bowels freely opened, pupils not dilated, pulse 88. During the next two days the headache was slight, the tongue much furred, and patient vomited once with the cough.

On the 15th he was observed by my then clinical assistant, Dr. Blackader, to be "very nervous, with much twitching of the limbs." Pulse 84. I saw the patient the next day at 5 p.m., and found the pulse 72, and regular; there was some twitching of the flexor tendons, principally at the wrists. The manner of the patient was somewhat confused, and he complained of weakness and giddiness. There was now no doubt as to the diagnosis. On the 17th he became drowsy, the pupils were contracted, the left more so than the right. The headache was mainly frontal, the patient had some difficulty in selecting words to answer questions. A small dose of calomel had freely acted upon the bowels. Temp. 99.1°, pulse 60, respirations, 18, slightly irregular. On the morning of the 18th, the respirations, 24 in the minute, were noted as being irregular both in frequency and depth. Pulse 60, temp. 99.8°. Patient had been sick three times in the night, and again this morning. The twitchings of the limbs, which hád considerably lessened, came on somewhat severely for half-an-hour this morning, but did not amount to convulsion. Evening temp. 99°, pulse 78, respirations 20. Jan. 19th, temp. 99.6°, pulse 84, respirations 26, more regular. Headache slight. On the 20th the morning temperature was 101.2°, pulse 94, respirations 26. Twitchings well-marked. Bowels confined. In the evening, temp. 99.6°, pulse 78, respirations 36. Patient passed a sleepless night and was dull and drowsy the next morning, the 21st. In the evening the temp. was 101.4°, pulse 92, respirations 30, somewhat irregular. He remained drowsy through the next day, increasingly so towards the evening. The temperature continued a little above 101°, pulse 93, regular, respirations 26. On the 23rd the morning temp. was 101.2°, pulse 90, respirations 30. At 3 p.m. patient was "drowsy looking, but intelligent, complaining of headache; some hesitation in answering questions. Heart's action regular, tongue protruded evenly, no paralysis of face." No paralysis of limbs. Retina examined by Dr. Blackader with no positive result.

Jan. 24th, some irregularity of the pulse noted early in the morning, temp. 99.4,°, respirations 30. Next day (25th) at 10 a.m., stertorous breathing came on suddenly, patient aroused with difficulty, eyes opened when spoken to, no other sign of consciousness, temp. 101°, pulse 72, respirations 36. At 3 p.m. "respirations 28, irregular in depth; pulse 96, weak, regular, slight subsultus, patient difficult to rouse, but complains of thirst." Evening temp. 101.4°, respirations 32, pulse 100, weak, but fairly regular, increasing insensibility. During the 26th patient was restless, appearing conscious at long intervals for a few moments only, urine passed under him, took nourishment with difficulty, temp. 100°, pulse 120, respirations 38. On the following day, both pulse and respiration became irregular, insensibility complete; at 10 p.m., marked lividity of face, pulse 138, respirations 40. Patient died at 2.15 a.m. on the 28th January.

The insidiousness of the onset of meningitis was well illustrated in this case. The symptoms were no doubt somewhat masked by the effect of the hyoscyamine. The furred tongue rendered the headache and slight vomiting less noteworthy. The muscular twitchings which occurred on the fifth day rendered the diagnosis certain, and they continued more or less throughout the illness. The memory of the patient was markedly impaired, and he had a difficulty in finding words to answer questions which he evidently understood. The temperature was misleading, as I have ever found it to be in tubercular meningitis; up to the 12th day it was lower than it had been the few days previously, and it never rose above 101.5°. The pulse was moderately quick, and was on one or two occasions irregular. The respirations were at times remarkably irregular, both in depth and frequency. Irregularity in respiration and in pulse, the two having no definite relation to one another, are common and important, but usually transient, symptoms in tubercular meningitis. I have never, however, observed anything approaching to the Cheyne-Stokes type of respiration in this disease.

Increasing drowsiness (with brief intervals of consciousness most difficult to explain) was in this as in all cases of tubercular meningitis the closing symptom, occurring intermittently at an early period, and gradually destroying consciousness entirely. Obstinate constipation, a common symptom in all head affections, was not in this case noticeable until the eighth or ninth day, when the diagnosis was well confirmed.

The following propositions[1] embrace what is reliable in the diagnosis of this complication, often so startling, at other times so insidious in its onset.

(1) *Persistent headache and vomiting are the most common first symptoms of the disease. They may or may not be combined. They are usually associated with furred tongue and disordered bowels, which tend to mask their significance.*

The headache of tubercular meningitis does not affect with constancy any particular portion of the head. It is sometimes frontal, often over the crown of the head, occasionally at the back or on one side. Although always a sign to cause anxiety when it occurs at all severely or persistently in phthisis, yet it is never, even when associated with vomiting, sufficient to enable us to form a diagnosis. In several cases of phthisis I have found headache so severe, persistent, and, taken together with the general aspect of the patient, so apparently characteristic of meningitis, as to have led me to feel confident as to its real significance—yet again and again my suspicions have proved to be unfounded. On the other hand, in the majority of cases of true tubercular meningitis that have come under my observation, there have been, as in the two cases above related, attendant phenomena to otherwise explain the headache until the appearance of more decided signs removed all doubt. Hence *headache or vomiting, although not sufficient for diagnosis, are signs which, if not readily relieved by treatment, should always arouse grave suspicions.*

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[1] The notes on which these conclusions are founded were included in a tabular form in a preceding edition of this work, page 161.

(2) *Disordered vision, impaired memory and confusion of ideas are signs which, taken in association with headache, are almost diagnostic. Muscular twitchings (and of course convulsions) absolutely so.*

Any or all of these signs may closely follow the appearance of headache or vomiting. They may, one or more of them, constitute the first symptoms of the disease. Paralysis of the third or sixth nerve usually occurs among the later symptoms, when the effused lymph and contractile tissue drags and exercises pressure upon the nerves. Ophthalmoscopic examination of the retina may yield a valuable positive result, but the absence of granulations upon the retina signifies little.

(3) *Drowsiness deepening into coma, but often with intervals of consciousness, is the most constant of the later symptoms of tubercular meningitis. It depends upon effusion into the ventricles.*

(4) *Irregular pulse, irregular respiration and excess of phosphates in the urine, are amongst the occasional signs of tubercular meningitis.*

Both irregularity of the pulse and of the respiration are not uncommon after the period of coma, but they are then signs of little importance. A marked irregularity of the pulse, occurring, however, early in the attack is I think of greater significance than is usually recognized. The pulse is commonly rather slow than quick, sometimes markedly infrequent. The respirations are rarely affected during the early periods of the attack.

(5) *The temperature is as a rule not much elevated in these cases of tubercular meningitis. It is more often raised towards the end of the attack, its rise being apparently associated with secondary inflammatory lesions. It is of little or no value in the diagnosis of tubercular meningitis.*

In the first place in cases of phthisis we have already a cause of possible elevation of temperature in the pulmonary disease present, and secondly, the temperature is not usually high in tubercular meningitis. On this point, however, the cases I have observed show that, contrary to the teaching of Wünderlich, the temperature rather tends to rise than to sink as the fatal termination draws near.

Excess of phosphates in the urine may be a valuable sign in helping us to interpret the earlier symptoms of tubercular meningitis. In one case already mentioned, it certainly led me to a right diagnosis some days before other absolutely certain signs presented themselves. There was no such excess present, however, in other cases,[1] so that this sign like some others will probably turn out to be of importance when present, but not essential.

The *duration* of this disease is various; it may terminate in a few hours, or days, or weeks. Patients will sometimes linger for many days in a state of complete insensibility from effusions into the ventricles.

LARDACEOUS DISEASES.—In about twenty per cent. of cases of phthisis, lardaceous changes are found in other organs. Thus from notes of 99 *post-mortem* examinations made consecutively at the Brompton Hospital, and excluding only a few which were imperfect, I found lardaceous degeneration present in 20 cases; 8 males, and 12 females. The pulmonary disease in these twenty cases had a maximum duration of 66 months, minimum 5 months, mean 26 months.

In eight out of the twenty cases death was caused by completeness of lung destruction, the lesions being caseous pneumonia in six, tuberculo-pneumonia in two cases. In all these cases the spleen was affected; twice in association with lardaceous, and twice with fatty, liver.

In *four* cases, one lung was mainly affected with cavities and pulmonary fibrosis. The other lung was enlarged, and contained some secondary tubercles. In three of these cases there was extensive ulceration of the intestines, and in the fourth case cystic degeneration of the kidneys and resulting uræmia. The spleen was affected in all; with lardaceous liver in one case, and with fatty liver in another. In *eight* cases the disease of the lungs was extensive and double, but yet not fully completed, the patients being cut off by associated lesions, *viz.*, ulcerated intestines six cases, the kidneys being also fatty in three: deep ulceration of larynx one case, and fatal hæmoptysis one case. The pulmonary lesions were pneumonic in five instances, also tubercular in three. The spleen was lardaceous in seven cases, in four in association with lardaceous liver, in one of which the kidneys and intestines were similarly affected; in two cases in association with fatty liver. The liver was alone affected in one instance. In the above statistics it should be mentioned that I am alluding to the coarser degrees of lardaceous change; in a considerably larger percentage the iodine test applied to fine sections would detect a trace of amyloid change.

It is impossible satisfactorily to explain the occurrence of amyloid change in this large proportion of cases of phthisis. It may be said generally that it occurs in the more chronic forms, and it is possible that long periods of quiescence, during which there is little or no pyrexia, are favorable to its development. It can scarcely be doubted that depuration is more or less directly the cause of the change, but there must be other factors of which we are not as yet informed.

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[1] I am here referring to the rude but facile test of obtaining a cloud on boiling which clears up on the addition of acid. Perhaps a more elaborate chemical analysis might be useful in such cases, but it could be only carried out in the laboratory.

The above cases were not investigated with reference to syphilitic history, but no syphilitic lesions were recognized amongst them. In cases, however, of syphilis, in association with phthisis, lardaceous disease of liver and kidneys is, in my experience, relatively much more common than in ordinary phthisis. Lardaceous disease may be recognized as a complication in phthisis by the detection of a large, smooth, hard liver, with well-defined and rigid marginal outline. Still more commonly the spleen may be found enlarged so as to present below the cartilages firm and smooth on palpation. Albuminuria considerable in amount, with hyaline casts, and a variable degree of dropsy, may be present.

It only rarely happens and chiefly in the fibroid type of phthisis, that the lardaceous disease causes death, and in these cases the fatal result is due to kidney complication. In a few instances the intestinal mucous membrane is involved, and obstinate diarrhœa exhausts the patient.

As a rule, however, the appearance of signs of lardaceous changes in other organs, suggests the probability that the case will prove one of more than average duration.

ALBUMINURIA.—The occurrence of albuminuria in phthisis is attributable to three forms of kidney lesions, *viz.*, lardaceous degeneration, scrofulous disease and a form of nephritis.

Lardaceous degeneration of the kidneys in phthisis is not common, much less so than a similar affection of either the spleen or liver, and when it occurs it is usually in association with a similar affection of the other organs. It is most commonly seen in the later stages of the most chronic fibroid forms of phthisis.

In the distinctly syphilitic forms of phthisis lardaceous kidney is more frequently met with, and at an earlier period of the disease. A considerable degree of renal dropsy may attend the albuminuria arising from this cause, and the abdomen is generally enlarged from the presence of an amyloid or amylo-fatty liver. Diarrhœa is often a marked symptom in phthisical albuminuria, and sometimes uræmic vomiting and convulsions occur.

A more common cause of albuminuria and renal phenomena in phthisis, in my experience, is a form of nephritis in which the kidney is somewhat enlarged, its capsule adherent, the organ softer than natural and less dry on section, presenting a swollen cortex of a mottled appearance from points and streaks of fatty degeneration. This kidney does not give the amyloid reaction. The albuminuria attendant upon this form of kidney disease is considerable in amount, there is a tolerably copious sediment from the urine, containing abundant epithelial and fatty casts.

This form of kidney disease occurs at an earlier period of phthisis than the lardaceous, and is attended with more decided symptoms. Indeed, whereas it is quite exceptional for the lardaceous kidney importantly to curtail the duration of phthisis, this form of kidney disease is very fatal, being attended with scantiness or suppression of urine, obstinate and exhausting vomiting or diarrhœa or pulmonary œdema and asthmatic phenomena, and other grave uræmic symptoms.

The pathology of this form of kidney disease has not been much inquired into; it would seem to be a slow nephritis with early degeneration of the renal elements.

FISTULA.—Fistula in ano occurs in something less than five per cent. of cases of phthisis, and in my experience is almost limited to males. Amongst those afflicted with fistula, however, phthisis commonly supervenes or already exists. There would seem to be two circumstances important to bear in mind with reference to this disease, *viz.*:—1. That fistula may arise from several causes, having no necessary connection with tubercle, but that having become chronic it is very apt to become tubercular, and to lead to secondary tubercle in other organs, especially the lungs. Characteristic bacilli have been found in the indurated walls of such chronic fistulæ. In cases of this kind timely operative treatment of the fistula may avert the secondary tubercular affection of its wall and of other organs.

2. It is more common for fistula to occur in the course of phthisis when the cachexia of that disease is already marked. It is impossible to say whether in these cases the disease is tubercular from the first, but it soon becomes so. In the presence of decided chest disease fistula becomes of secondary importance, and unless causing much additional suffering and exhaustion, it should not be interfered with by operation. There is sometimes to be observed a kind of alternation between the activity of the fistula and that of the chest disease, and this has deterred surgeons from operating with willingness; but a more real objection is the risk of the operation proving a failure from the morbid character of the surrounding tissues, and the further danger of fresh activity of chest mischief supervening upon the breakage of the tubercular induration about the fistula, or upon the renewed febrile action attendant upon operations in unhealthy tissues.

There are cases, however, in which the local inconvenience and suffering from the fistula are so great as to necessitate some operative measures.

CHAPTER XXIX.

GENERAL REMARKS ON THE PROPHYLAXIS AND TREATMENT OF PHTHISIS.

THE prophylaxis of phthisis, generally speaking, does not essentially differ from that of other diseases; all measures that tend to invigorate, also diminish mortality from consumption as from other constitutional maladies. On glancing through the chapter on etiology, however, it will be seen that there are certain conditions which favor the occurrence of phthisis more than other diseases, and that the tendency to the disease is inherited in a large proportion of cases. In avoiding the pernicious influences of these conditions, especially over-crowding, sedentary employment, dusty occupations, etc., and by taking extra precautions in all cases of hereditary predisposition, we do the best that can be done, generally speaking, in the way of prophylaxis.

It is obviously undesirable that persons with a marked phthisical taint should marry, and still less desirable that they should intermarry; the advice of the physician in these matters is, however, not commonly followed by the persons most immediately concerned.

All measures calculated to avert the possibility of contagion are measures of simple hygiene.

Threatened and incipient phthisis.—The child of a consumptive mother should be weaned early, or should have a carefully selected wet-nurse. If the alternative of a wet-nurse be not adopted, however, there can be little doubt that the child will stand a much better chance of being reared if it be suckled by its mother for the first month of life, than if brought up wholly by hand. If a healthy foster-mother be provided, the child may be suckled entirely, until eight or nine months old.

The hygiene of the nursery must be very strictly looked to, especially with regard to the avoidance of dust, the free admission of air without draughts, the provision of a separate cot or bed for the child, and abundance of air space in the night nursery. Milk must form a large item of the dietary throughout childhood, the other elements, saccharine, farinaceous, nitrogenous, and saline, being duly provided. Careful attention to the skin, by the use of a tepid bath at least once a day, warm, light, all-wool under-clothing, loosely fitting and made to cover the chest to above the clavicles, and abundant out-door exercise, are the principal means of securing sound health. The plan of keeping delicate young children with naked legs, arms and upper chest, with the view of hardening them, is too obviously absurd to need further comment.

At the period, about the age of seven, when children begin to go to school, the question comes whether a day-school or boarding-school shall be selected, and in most instances it is decidedly preferable for delicate boys, and perhaps girls, to be sent away from home to a carefully managed preparatory school, than for them to attend a day-school from their own homes. The divided responsibility in matters of hygiene, food, clothing, and the like, the hurried meals, irregularity in exercise, and increased exposure to cold and wet in bad weather, which attendance at day-schools involves, are full of risks. There is also no doubt that delicate children, from the extra solicitude which has been necessary in rearing them, become whimsical and morbidly self-conscious at about this age, and the regularity and discipline of school life is good for them. The school should be well situated in the country, on a well-drained soil at the seaside, or in the neighborhood of the sea. The modified sea climate of Bournemouth suits children well, also the high ground of St. Leonards, Ramsgate, and many other places. The experiment of sending delicate children to the Swiss mountain resorts, Davos, St. Moritz, etc., has perhaps hardly been sufficiently tried, their subsequent well-being on leaving the mountains not having been as yet fully tested.

Whooping-cough and measles are antecedent to a large proportion of cases of phthisis in children, and the utmost care should be taken to ensure complete convalescence from these diseases.

An undue irritability of the lymphatic glandular system is often to be observed in children prone to phthisis, and the glands when enlarged tend to become caseous, remaining, as it were, magazines of tubercular poison. Hence all sources of gland irritation, decayed teeth, eczemas, eruptions on the scalp, catarrhal affections of the bowels and bronchi, should be carefully and promptly treated. Chicken-pox in children often leaves behind troublesome sores about the head and body, which are very likely to lead to glandular enlargements, and the utmost care should be taken during this disease to protect from the air by means of collodion, all pustules which are large and likely to ulcerate. In cases in which glands become caseous and suppurate, it is better to scrape out the gland so as to remove at once all caseous matter than simply to incise it; less scarring is thus caused, and the danger of infection of other glands by retained caseous matter is removed. Short courses of cod-liver oil and steel wine or Parrish's food should be given to delicate children, extending over three or four weeks, at intervals during the winter and spring, and especially after the occasional catarrhs to which they are all liable.

There is some foundation in experience for regarding the succeeding periods of seven years as critical in matters of health; there are certainly grouped about the first and second dentition, and the periods of adolescence and manhood, developmental changes and associated external circumstances of life, that favor the occurrence of certain diseases, and peculiarly of phthisis.

Between the ages of 15 and 21, phthisis is very liable to develop in those predisposed, and an opportunity may often be taken after the completion of school education, or at the end of college career, to secure a period of six or twelve months to be devoted to the re-establishment of sound health. Many plans may be devised with this view, suited to the circumstances and means of the patients and their friends. A long sea voyage with a responsible companion is one of the best measures, or twelve months' residence on a farm in a healthy part of this country or abroad. With girls it is more easy to arrange a series of visits to healthy parts of the country and abroad, where an out-door life can be to a great extent secured. The future profession or business of a youth may in the course of these years be determined upon, sedentary pursuits being avoided, and those encouraged which are associated with an active outdoor life. There are some cases, however, and they are not very uncommon, in which with phthisical predisposition the mental faculties are keen, whilst the bodily conformation is not such as to withstand, or respond to, a rough physical life. In such cases a sheltered life is to be recommended, with such pursuits as the individual is best qualified for, and with such precautions in the way of hygiene, exercise, and climatic change, as may be possible, and best adapted to the case. The experienced physician can recognize in many such cases that the fund of vitality is small, and that to attempt to lay it out on an ambitious scale, with a view to large or long-continued returns, is to risk the loss of the whole.

"Neglected colds" enter into the history of a large proportion of cases of phthisis. The best routine treatment of an acute catarrh during the first twenty-four hours consists of full doses of acetate of ammonia or citrate of potash, frequent fomentation of the nasal passages with weakly carbolized water, two per cent., with hot foot-bath and a little Dover's powder at bed-time. In two days quinine or chloride of ammonium may be commenced with, the patient still remaining indoors. After about the third or fourth day, when all febrile symptoms have subsided, a week's change to some accessible sea-side place will commonly cure the catarrh. Sometimes at the first onset of the malady a full dose of quinine will arrest it; every effort must be made to prevent a catarrh from lingering or becoming chronic. In all cases of acute inflammatory chest affection in patients with phthisical tendency, the utmost care should be taken to ensure complete recovery, and it often takes a long time thoroughly to effect this. It is well after any such attacks to spend the ensuing winter in taking a voyage, or at a health resort adapted to the circumstances and case.

General remarks on the treatment of phthisis.—There is much in the treatment of phthisis which is in common with the treatment of other diseases. There are three points, however, which may be especially borne in mind with regard to pulmonary disease, *viz.*—1. That in the respiratory process samples of the surrounding air are constantly being brought in contact with the extended respiratory surface, some portions of which are, in consequence of disease, lacerated, suppurating, or bathed in morbid material ready to decompose and abounding in specific germs.

The remembrance of the facts embodied in this statement is enough to emphasize most strongly the importance of all hygienic measures calculated to keep the air pure and free from organic and inorganic dust, and the necessity of abundant cubic space being allotted to such invalids, perhaps even beyond the necessities of others.

2. All the blood of the body passes through the lungs, the pulmonary circulation in this respect balancing the systemic circulation.

From this fact flow two or three considerations in the treatment of phthisis.

a. Any conditions which hurry the general circulation cause an unduly proportionate stress of blood current through the lungs, and hence the importance, in active disease of these organs, of muscular and mental rest, or at least quietude.

b. In chronic pulmonary lesions in which the disease, having effected a certain measure of destruction, is stayed, and the patient is regaining strength, flesh, and color, a point is not infrequently attained when there arises a relative systemic plethora, the blood volume and systemic metabolism out-balancing the vascular and functional capacity of the lungs. Fresh pulmonary hemorrhage, congestions, dyspepsias, diarrhœa, are the natural consequences which tend to rectify this perverted balance, but which once started, rarely stop within salutary bounds. Timely moderation in tonics and reconsideration of dietary and exercise, will avert such disasters.

3. Considerable tracts of lung are in health held in reserve for temporary service on occasions of unwonted exertion.

It is the development and bringing into daily action of such reserves, that constitutes a most important element in arrest of, or "recovery" from, phthisis. This development can be encouraged at the fitting time by graduated exercise on the incline, by residence in high climates, or by both combined, perhaps also by pneumatometry. Mere expansion of lung, however, be it remembered, does not constitute compensatory development; there must be also development of capillary circulation and good textural nutrition. The cautious stimulation of blood pressure and respiratory function gained by regulated exercise, most efficiently aid the natural tendency to the changes desired.

The *hygiene of the sick room* is of the utmost importance in the treatment of phthisis. The dwelling rooms of the consumptive patient should be of good size, lofty, and well ventilated, with a free exposure to the south or south-west, and sheltered from the north and east. The furniture should be sufficient for comfort, without superfluity. Carpets and curtains should be easily removable, so as to be shaken and dusted out of doors; all sweeping of carpets should be strictly prohibited in the invalid's rooms, and the floors and furniture should be kept free from dust, by the use of cloths rendered damp by Sanitas fluid or other weak solution of a cleansing kind. Washing-chintz coverings to the furniture are greatly to be commended; with two or three well-chosen patterns, the change from time to time is grateful and cheering to an invalid much confined to a suite of rooms.

Much irritation, cough, and increased activity of disease, will by these simple measures alone be avoided, and the more the patient is confined to his rooms, the more essential is it that such hygienic measures be strictly looked to. In the presence of broken and highly absorbent surfaces deprived of the means of rejecting harmful matters, the mechanical irritation of inert dust, and the septic influence of putrefactive and other organisms, are fertile of mischief. Sputum vessels should be used and should contain some Sanitas or thymol solution, or other disinfectant. Gas-lighting should be forbidden in the living rooms. The rooms must be maintained at an equable temperature, the sitting-room between 60° and 64°, the bed-room over 52.° The patient must have a separate bed, springy, with horse-hair mattress, not heavily curtained, and sufficiently but not heavily covered. In bed-ridden cases it is often a good plan to have two beds in occupation, so that a change may be made from one to the other. The clothing of the patient should be warm and light, and, even in the warmest season, thin woollen or silken garments should be worn next the skin.

The dietary of the phthisical patient must be framed on a very liberal scale, so as to contain a due share of animal and vegetable food and salts. The appetite and digestive powers of the patient are in many cases sufficient guides as to the amount of food to be taken, but the results of Debove's method of feeding phthisical patients artificially by means of an œsophageal tube, show what clinical observation also teaches, that appetite often fails when the system is nevertheless ready and able to assimilate much larger quantities of food. This fact is especially to be borne in mind during the hectic period of phthisis, when it is most important to sustain the patient by nourishment given in much larger quantities than inclination would prompt him to call for. On the other hand, at periods of continued pyrexia, it is desirable to simplify and restrict the dietary.

In cases of quiescent phthisis the natural appetite returns, and patients as a rule take food with avidity. Sometimes when flesh and blood are being rapidly made, and a tendency is observed for the body weight to get beyond the lung capacity, it is advisable to restrict the dietary somewhat, by diminishing malt liquors, substituting fish for butcher's meat, and suggesting some restrictions in the amount of fluids and solids taken. Coated tongue, quickened pulse and respiration, restlessness, dyspepsia, and increased breathlessness on effort, will, in the absence of any fresh lesion, lead to the recognition of this condition already referred to as one of *relative plethora.*

With these preliminary remarks I cannot do better than quote the general plan of dietary suggested by Dr. Weber in his recent admirable lectures:

"I have mentioned before that, in the majority of ordinary cases of phthisis, except the very chronic and arrested forms, it is better to take the desirable amount of food in frequent smaller meals than in two or three larger ones. I am in the habit of recommending a plan of the following kind, with many modifications according to circumstances.

"At 7 o'clock, or earlier, while still in bed, a cup of milk, with a dessert or tablespoonful of cognac, or with lime-water, or with a small quantity of tea or cocoa, and a small piece of bread and butter.

"At half-past 8 or 9, after dressing, breakfast of milk, with some slightly stimulating addition, as tea, coffee, or cocoa, bread and butter, or bacon or ham, or fish.

"At 11, a tumblerful of milk or koumiss, or sometimes a cup of broth or beef-tea, or a sandwich and a glass of wine.

"At 1 or 1.30, a substantial meal of meat or poultry, or fish, or game, with fresh vegetables, some light pudding or cooked fruit, and a glass of wine.

"At 4 o'clock, a glass of milk or koumiss, or a cup of tea or coffee with much milk, and some bread and butter or plain biscuit.

"At 7 p.m., another substantial meal, similar to that in the middle of the day.

"At 9.30 or 10 p.m., on going to bed, a cup of milk, or bread and milk, or milk with some farinaceous food, as Hart's or Liebig's or Nestlé's, or Mellin's. At this time, if there be night-sweats, the addition of a tablespoonful of brandy is very useful.

"In cases of considerable pyrexia, it would be injudicious and impossible to give as much solid food as in chronic non-febrile, or nearly non-febrile, cases; but it is necessary to give as much easily digestible food as the patient can digest. Our aim ought to be somewhat to check the waste, and to replace by food the increased waste. Here alcohol is of great use. Milk is often not digested in its natural state, but it must be tried peptonized, and diluted with pure water, aërated water, and still better, with barley water, or thin gruel. Chicken-broth, veal-broth, beef-tea, and gelatinous substances are, in these conditions, most useful; while, in health, and in the non-febrile consumptive cases, they ought not to take the place of the more solid proteinaceous substances.

"In most cases of phthisis, it is desirable to introduce into the system a fair amount of fat, and this can often be done better in the shape of bacon, fresh butter, and milk and suet, than in the form of cod-liver oil, though the latter too is most useful."

Certain special kinds of diet have from time to time been suggested as curative of phthisis, such as the *milk-cure*, the *whey-cure*, *koumiss-treatment*, the *grape-cure*, and the like.

None of these measures of treatment, however, will as "cures" bear examination; much of the benefit of the treatments being attributable to the healthy surroundings of the "cure," and to the admitted fact that such cures are only adapted to a limited number of favorable cases of the disease.

This limited number of favorable cases of phthisis, it must be indeed confessed, make the reputation of every health resort and "cure" in turn, and attract to them cases for which they are not adapted.

The grape-cure is especially carried on at Méran, Montreux and some other resorts in Europe, from two to six or more pounds of grapes being consumed in the twenty-four hours. Professor Lebert[1] recommends half a pound of grapes to be taken in the early morning, 7 a.m., and again at 5 p.m., and, after a few days, a third quantity at 11 a.m.; a further gradual increase to a pound on each occasion, in those cases in which the fruit is well-borne, being the safe limit to which this treatment can be carried in phthisis. The diet at other meals must be light, digestible, and unstimulating.[2] There is no doubt that cases of phthisis are frequently not allowed sufficient vegetable food and salts in their dietary, and when and where grapes are in season the substitution of them for the intermediate meals may often be of value, especially in cases of hectic associated with torpidity of bowels. After each meal of grapes Lebert recommends patients to rinse the mouth with a little soda or potash water. September and October are the grape-cure seasons.

The exclusive use of milk is not adapted for the treatment of any form of consumption, but in all cases, and especially in young subjects, milk to the extent of from one to two pints may be taken daily by those who can digest it. Cows' milk (new or skimmed), asses' milk, goats', mares', or fermented mares' milk (Koumiss) may be used. In some cases whey may be preferred, and for those who cannot take other forms of milk it should be tried, especially in cases in which there is a considerable loss of salts in night perspirations.

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[1] Quoted by Dr. B. Yeo, in chapter on The grape-cure, in his work on Climate and Health Resorts, p. 313.

[2] See also Von Ziemssen's Handbook of General Therapeutics, vol. i., On the Dietary of the Sick and Dietetic Method of Treatment, by Professor Bauer, translated by Dr. E. F. Willoughby, 1885, p. 331.

Koumiss,[1] the fermented milk of mares, has from all time been used as a beverage by the inhabitants of the steppes of Southern Russia, the best koumiss being prepared from pasture-fed mares which have not been put to work. Ssamara is the steppe district where Russian koumiss of the best kind is made, and the best quality is obtained in May, June and July, when the climate of Ssamara is said to be very beneficial. Dr. Karrik, one of the resident physicians, speaks well of the clear, dry, aromatic air of the summer at Ssamara as enabling the patient to drink largely of koumiss. A glass or two of koumiss is taken in the early morning, three or four glasses in the forenoon, and as many in the afternoon. No other drink should be taken, and no sweets or alcohol in any other form, the meals consisting of a liberal allowance of mutton, poultry, eggs, butter and bread. The treatment should be commenced with caution, and should extend over a period of two or three months; as much time as possible being spent in the open air riding or walking. Drs. Postnikoff's and Annaeff's are the principal establishments at Ssamara for carrying out the koumiss-cure, and it is only to such establishments that patients could be safely sent for treatment, although a more robust and enterprising invalid might on a second visit venture upon a more independent and nomadic life in the country. The koumiss-treatment is best adapted for cases of chronic bronchial catarrh, incipient, quiescent, and very chronic phthisis. Cases of extensive disease or in the active pyrexial stages are manifestly unfitted. Such cases, however, may often with profit take a few glasses of koumiss daily between meals.

Of late years, since the introduction of koumiss into London by Dr. Jagulski, this beverage has been largely used, and it is prepared by the Aylesbury Dairy Company in London. For those who are beyond the reach of this source of supply, the following formula, which has been kindly supplied me by my friend Dr. Charles of Cannes, who uses the preparation largely in the South of France, may be found useful.

Home-made Koumiss.—Nearly fill a quart bottle with fresh milk, leaving enough room to shake it up easily; add a spoonful of crushed lump sugar; and a bit of German yeast[2] the size of two ordinary five-grain pills; cork and tie down with wire or string; keep in a cool place and shake twice a day. The koumiss will be ready to drink on the sixth day in average weather. Earlier in hot, and later in cold weather. A thinner koumiss is made from skimmed milk. This more resembles the koumiss made from mares' milk. Most people can digest that made from unskimmed, and *for them* it is a mistake to use skimmed milk. All the bottles, corks, etc., must be scrupulously clean.

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[1] For authorities and for further details, the reader is referred to the appendix on Koumiss-cures, by Dr. Stange in Ziemssen's Therapeutics, op. cit., 343, et. seq.

[2] The German yeast is to be got from any baker who makes Vienna or fancy bread. It putrefies and must therefore be used fresh. If kept freely exposed to air it dries up and becomes inactive. If kept corked up in a bottle it is apt to putrefy. It will keep a week if placed in a cup loosely covered up with paper.

The digestive system of phthisical patients requires very careful attention, and in many cases the whole treatment consists in establishing a working equilibrium between the digestive powers on the one hand and the quantity and quality of food taken on the other; the aid of medicines being called in to support digestive powers and to correct digestive failure. In detail this treatment is based upon the ordinary principles of medical art, and ingenuity should be exercised to give as few doses of medicine as possible. Tonics are rarely needed more than twice in the day, and often once a day is sufficient. Cod-liver oil can be timed to be taken with one or both doses of tonic. Cough mixtures are as a rule better avoided altogether, or a single dose given at bed-time to secure rest. Night sweats may often be relieved without medicine.

A fertile source of dyspepsia is the swallowing of expectoration, and patients should be carefully warned of this.

Exercise must be taken in accordance with the patient's strength, and the activity and stage of the disease. In the active phases of the malady, with elevation of temperature, quick pulse, and hurried breathing, all symptoms will be intensified by exercise, and complete muscular rest must be enjoined, in association with the best air conditions that can be provided. By means of hammocks, tents, sheds, bath-chairs, moveable beds,[1] and appropriate arrangement of wraps, all the advantages of open air may be obtained without exercise in suitable climates and seasons; and much care in room hygiene on the lines already laid down will compensate for extra time spent indoors in consequence of bad weather. In early stages of quiescent cases an outdoor life is to be advised, and those occupations and climates selected in which this can be best attained.

There is perhaps no disease in which there is so large a demand for discretion in advising with respect to climate as in phthisis, and how difficult it is to be discreet yet not vacillating! In the premonitory stages the physician may speak firmly even imperatively, but his words are often vain to move patients to those prompt steps which alone can save them. The victims are on the threshold of life or still more commonly in the early stages of professional or commercial success; some cannot, others will not, make the necessary sacrifices; "the doctor may be wrong." Others again wait on till "to-morrow, and to-morrow, and to-morrow," whilst their disease "creeps in this petty pace from day to day," and by the time they have finally made up their minds to take the measures long since advised, the opportunity has passed; the measures fail, and the doctor is blamed.

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[1] One of Ward's, Aldermann's or Carter's mechanical bed-chairs is a great luxury as an addition to the sick-room furniture.

In the next stage of confirmed but early disease, advice cannot be given with the same earnestness; we must make compromise with surrounding circumstances, and endeavor to estimate the probable duration of the case, and how far it will be really influenced by the measures proposed, whether the sacrifices involved to the patient or his family may not be too great for the results to be hoped for. The tendency at this period is to shirk the responsibility of *ordering*, and to attempt to guide for the best the patient's own inclination.

In the later stages of the disease, however, it is often the patient or his friends who are all eagerness for a move. They realize that the lungs are "touched," that "they must give up for a time" in order to be restored. They are ready to venture their last remnant of strength, their last sixpence of resource, on what the physician knows to be a hopeless quest. Let me say at once of this latter group of cases, that as a rule they are best at home, wherever that home may be. Such patients should not be advised to leave home unless they can take their responsible friends or families with them, and can secure all the physical comforts and mental quietude that they would have in their own homes. A small proportion of persons with advanced phthisis can afford to transplant themselves and their families with every luxury. The majority are wisely advised to spend a fraction of the proposed outlay in making their own homes better adapted to their wants.

I will content myself with regard to climate by mentioning those which are best adapted to each of the chief varieties of phthisis, in conjunction with other points in special therapeutics. In the works of Archibald Smith, Braun, Marcet, Williams, Jaccoud, Burney Yeo, Sparks, Hermann Weber, and others, will be found details on the subject of climate, which cannot yet be considered as ripe for condensed and exact treatment.

CHAPTER XXX.

REMARKS ON CLIMATIC CHANGE AND TREATMENT FOR EARLY STAGE CASES OF PHTHISIS.

THE climatic resorts to which patients with early stage phthisis may be sent, differ with the time of year, the nature of the case, and the temperament of the patient.

It is impossible at the present moment to classify these resorts with exactness; there are, however, certain indications for selection generally applicable, which I will endeavor to point out.

The most momentous question which first presents itself for decision with respect to cases of early stage phthisis, is whether to send the patient to an elevated climate or not. It will clear the ground, therefore, if I first briefly consider these climates, and the cases that should and should not be sent to them.

The climatic stations most typical of the kind in question, are the Alpine stations of Davos (5000 ft.), St. Moritz (6000 ft.), Samaden (5000 ft.), Wiesen (4700 ft.); the two first named being at present the most important, and the list being yearly added to. It is during the winter months that the properties of these climates, which are regarded as remedial in phthisis, are especially to be found. These properties are: —1. Low atmospheric pressure, some five inches of mercury below the barometric measurement at the sea-level. 2. Dryness of atmosphere. 3. Purity of air, *i.e.*, freedom from organic and inorganic dust. 4. Antiseptic qualities by virtue of freedom from organic germs, and the relatively large proportion of ozone present. 5. Low temperature of the air. 6. Diaphaneity to the sun's rays, both illuminating and chemical, from the thinness and clearness of the atmosphere. 7. Stillness of the air, in consequence of which its coldness is less felt.

To the rarefaction of the air of these regions as a stimulant to the respiratory function, to its aseptic qualities by virtue of which putrefactive and fermentative processes are hindered, and to the vivifying influence of the brilliant and warm sunshine which enables patients to be much out of doors during six hours of the day, their beneficial effects are attributed.

The diaphaneity of the rarefied atmosphere of these high regions is important in moderating the effects of the enormous difference between sun and shade temperatures, and in rendering the diurnal range of temperature much more equable than would be at first sight apparent. Dr.

Denison and Dr. Weber[1] have both drawn attention to this point, *viz.*, that the air itself by virtue of its diathermic properties is very little warmed by the sun's rays, which nevertheless so powerfully affect objects exposed to them. Dr. Denison puts the matter shortly by giving the following rule of increasing diathermancy of air, *viz.*:—"For each thousand feet rise in elevation there are about four degrees greater difference between the temperatures in the sun and in the shade, on perfectly clear days at 2 p.m., as recorded by the black metallic-backed thermometers, other influences than those of sun and shade being excluded."[2]

Improved sanguification and nutrition, expansion of unaffected parts of the lungs, and restriction of the limits of diseased areas, are results obtained in a very striking degree in successful cases by residence in these localities. They are, however, rigorous climates, and it must not be forgotten that potent for good in well chosen cases, they are also active in working mischief to those whose condition is not well adapted for them. A great advantage pertaining to these resorts is the existence of large, well-ventilated, residential establishments, where the patients are more or less under the direct guidance and control of experienced physicians. The comparative isolation from the surrounding world, which is a necessary condition of residence in these parts during the winter months, secures this advantage of superior medical control, although in another respect it has a depressing influence by bringing into close relationship a number of people, the majority of whom are afflicted with the same malady.

Careful observation impels me to the belief that in one respect the curative influence of elevated climates has been greatly exaggerated; in regard, namely, to their having any exclusive power by virtue of rarefaction of atmosphere, of developing healthy lung and contracting diseased areas of lung.

I have often observed the development of unaffected lung proceed to the utmost possible limits in patients who have never been at a higher level than the galleries of the Brompton Hospital. Amongst dwellers in towns or country patients attending the out-patient departments, and in private, I have met with a fair proportion of cases which have recovered in the most different places, and under the most diverse circumstances, *e.g.*, in Yorkshire, Ventnor, Bournemouth, the Riviera. The physical state common to all such cases is contraction of the limits of disease, and compensatory enlargement of the opposite and of the free portions of the same lung. I doubt the possibility of exceeding the compensatory development that may be observed in these favorable cases of arrested phthisis in the plains. The question is whether a larger number of such cases are to be observed in the mountains. This has not yet been shown, although it may well be expected. I am only desirous to point out that rarity of atmosphere, although it may be an auxiliary, is not an essential, nor in any sense the most important, condition for the bringing about of such development.

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[1] Croonian Lectures, 1885.

[2] The Rocky Mountain Health Resorts, 1882, p. 66. Also Analysis of Atmospheric Humidities in the United States, 1884, Chicago, p. 19.

Dr. Weber observes that "the compression of the diseased parts through healthy and emphysematous dilatation of the surrounding tissue," is one of the influences leading to arrest of the disease. But in all cases of arrested phthisis that I have seen, immediately around old indurated nodules, emphysematous dilatation of pulmonary tissue may be noticed, and this has always appeared to me a mechanical consequence of collateral aspiration to, aided by impaired nutrition of, adjacent air vesicles; nor can I understand the mechanism by which diseased areas could be compressed by surrounding emphysema, although they might, to clinical observation, be somewhat obscured by it. I have elsewhere pointed out the importance of distinguishing between merely enlarged or dilated lung and truly hypertrophied lung, in association with localized or arrested disease of some other portion. There is no advantage, but much the contrary, in producing emphysema in phthisis.

The cases suitable for treatment at elevated resorts are those of the early catarrhal and quiescent pneumonic group, especially in persons of lymphatic temperament with a moderate degree of anæmia. Cases of threatened phthisis from hereditary tendency, defective chest conformation and capacity, incomplete recovery from acute or sub-acute inflammatory affections, especially pleuritic and parenchymatous, are peculiarly adapted for such climatic treatment. Cases of more advanced disease, provided it be restricted and inactive, are also, as will be presently pointed out, suitable. It is important that the reserve lung be sound, not emphysematous, and that the heart and vessels be also healthy. It is quite a separate question whether some cases of cardiac disease would not do well at these elevations.

Cases which have commenced with hæmoptysis, and in the course of which hæmoptysis has from time to time occurred from active or passing congestions, are not thereby disqualified for residence in high climates, but a few weeks should elapse before removal there. For cases of recurrent hemorrhage, however, in which an ectasia or aneurism of a pulmonary vessel in a localized cavity is suspected, such climates would not be entertained. The following conditions are unsuitable for residence in high climates, viz.:—The erethric[1] constitution in any stage of the disease; the acute stage of phthisis in any form; with rare exceptions, chronic or sub-acute tubercular phthisis; advanced phthisis; phthisis complicated with marked emphysema, albuminuria, laryngitis or intestinal ulceration;

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[1] Nervous, irritable subjects, neuralgic, dyspeptic, bad sleepers, with habitually quick pulse and dry, harsh skins.

senile phthisis (as a rule); phthisis associated with general bronchial irritation partaking somewhat of the character of scattered broncho-pneumonia or peri-bronchial tubercle.

The winter season from late November to March is the best to choose for residence in these localities, but in those cases in which benefit is experienced, continued residence through the summer at about the same elevation, and a second winter in the same locality should be decidedly advised. A third or fourth year may sometimes be thus well-spent. In cases of mere delicacy or threatened disease during convalescence from some acute chest malady, a single, short period of residence may suffice. It is a question to be yet worked out how far in actual disease a short residence at a mountain resort will better fortify against a relapse on return to work in the plains, than some other measures, as a voyage or sojourn elsewhere; for in this practical world but few people can afford to live indefinitely in idleness above the clouds.

The spring and late autumn are the worst periods of the year for residence in the Alpine resorts. For those already in residence there, however, the spring is a dangerous time for travelling, and it is perhaps better to face it out with extra precautions.

Dr. Hermann Weber advises that patients should if possible get out to their proposed winter quarters on high ground by the end of August or early in September, in order to get acclimatized before the uncertain weather with which winter is ushered in, commences. It is certainly hazardous when the severe weather has set in to send delicate people from the plains to face at once the great change in atmospheric pressure and in temperature, and only in exceptional cases can this double experiment be advised. Dr. Jaccoud is very emphatic on the importance of making the ascent gradually, resting for a week or ten days at several intermediate stations. Such places as Ragatz (1700 ft.), Chur (2000 ft.), Promontogno (2600 ft.), Seewis (3000 ft.), Wiesen, at any of which good accommodation can be bespoken, might be chosen as stepping-stones to the highest points, and as convenient places for resting on the way down. Promontogno and Seewis[1] are good spring places for those who wish to keep on fairly high ground.

Among the elevated resorts in North America may be especially mentioned the Rocky Mountain stations[2] of Manitou, Colorado, Denver and Yellow-stone[3] situated between 5000 ft. and 6000 ft. elevation, and resembling the Swiss resorts in general features. In the Peruvian Andes,

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[1] At the smallest stations mentioned, and indeed at all the Alpine stations, it is very important to secure beforehand the desired accommodation. Seewis is well-spoken of by Burney Yeo. op. cit., p. 300, and H. Weber.

[2] Denison's Rocky Mountain Health Resorts, also his Climatic Maps of the United States, 1885.

[3] Hermann Weber, Croonian Lectures, 1885, p. 107.

Jauja, Santa Fé de Bogota, and Quito, at an elevation of about 10,000 ft., the effects of a highly rarefied air are obtained with a temperate and a brilliant climate. The South African Highlands of the Orange States, although possessed of a climate perhaps superior to any of the others, are as yet unsuited for invalids, except those who are experienced travellers and sufficiently robust to be trusted to follow their own devices. The Indian hill stations are by no means equal to the others mentioned for the treatment of phthisical cases, although good accommodation can be obtained at most of them. At Darjeeling, near Calcutta, a very fine sanitarium has been recently built by the government of India for the reception of invalided officials in three grades; but like the other hill stations it is adapted for spring, summer, and autumn, not for winter residence.

There are several places of lower elevation, amongst which Görbersdorf in Silesia (1800 ft.), Falkenstein in S. W. Germany (1600 ft.), Méran in the Austrian Tyrol, Lausanne, Glion, Les Avants, may be mentioned; at the first three of which there are establishments where the hygienic treatment of phthisis is carefully supervised by skiilful physicians. In these places the element of a rarefied atmosphere is wanting, but the air is clear, dry, and bracing, and is adapted for much the same kind of cases as the higher resorts. Dr. Brehmer of Görbersdorf was one of the first to advocate an open-air and bracing treatment of the disease, and at his establishment, and that of Dr. Dettweiler of Falkenstein, terraces are arranged to form inclined ascents for graduated exercise with a view of developing the respiratory powers. At Méran the "grape cure" is conducted in the autumn, the "Koumiss cure" in the spring, and baths and inhalations all the year round.

Notwithstanding the enthusiasm with which cold mountain climates have been of late years advocated for the treatment of consumption, it remains certain that these resorts are not adapted for the majority of patients, as they come before us, suffering from this disease; and perhaps it might with truth be said that a large number of the very cases that do well aloft, do equally well in the plains.[1] It is indeed only by weighing all the circumstances of each individual case that it can be decided whether on the whole a better result may be hoped for by a sojourn at the higher or the lower stations, or by a sea voyage. A glance at the long list of cases in which the choice of an elevated resort would be positively harmful, will suffice to show the importance of a careful diagnosis being made as the first step towards a solution of the problem.

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[1] A curious illustration of this remark will be found in the fact that M. Jaccoud, op. cit., p. 371, most strongly recommends the climate of Madeira as the very best that can be selected for cases of quiescent pneumonic phthisis; the very cases that most authors would advocate for treatment in the high and dry Alpine resorts.

Perhaps such practical remarks as I have to offer with regard to the selection of marine and maritime climates, and inland climates of the plains, may be best included under the seasonal headings which conveniently conform with the want of patients.

MARINE, MARITIME AND INLAND CLIMATES. *Autumn.*—(September, October, November). At this time of the year the marine and maritime climates are especially suitable, and during the latter part of the season, when the leaves are falling and rotting on the ground, the inland country districts, wherever there are many trees, are eminently unsuitable for cases of chest disease or delicacy. Extensive pine districts form an exception to this general rule. During the first two-thirds of the autumn season the moorland districts of Scotland, Yorkshire, and Devonshire, and such dry localities as Malvern and Tunbridge-Wells, are also well adapted for pulmonary cases.

Sea-side resorts are characterized by abundant air space, great purity of air, with relative excess of ozone and of moisture, and an equable temperature and great freedom from dust. As the season advances the temperature is also relatively warmer as well as more equable than in inland districts.

Most material advantages of popular sea-side resorts, however, are the abundant accommodation and variety of food they can afford to invalids, with a constant and inexhaustible reservoir of the purest air, and great facilities in the form of dry, level walks, sheltered seats, carriages, bath-chairs, etc., which are at hand. For the first half of the autumn season, Northern and East coast places on our own shores may be selected, Nairn, Scarborough, Clacton, Broadstairs, Margate, Cliftonville, Ramsgate, Folkestone, Ilfracombe. During the latter part, Brighton, Hastings, Bournemouth, Ventnor, Torquay, Tenby, Grange, etc. The more bracing places should be chosen for those with a scrofulous element, of lymphatic temperament, and with no marked tendency to laryngeal or bowel complications. In hemorrhagic cases, and those with larynx and bowel complications, the softer climates of Hastings, Ventnor, Torquay, and Tenby are as a rule preferable. In cases of nervous irritable temperaments, with disposition to neuralgia, sleeplessness, and torpidity of the liver, modified sea-climates, such as can be obtained a little inland at Bournemouth, Bexhill, Preston, St. Mary Church, etc., often agree better.

Of places on the Continent, Biarritz is an excellent autumn and early winter climate. Méran (Aust. Tyrol), and Montreux, Chexbres, and a few other Swiss resorts, may be advised, especially for those who desire to try the grape cure.

These latter places are convenient for those who contemplate the Riviera or Swiss mountains for the winter, and at either Méran or Montreux the stay may be prolonged into the winter months.

Sea voyages.—The latter part of the autumn, as soon as the equinoctial gales are over, is the best time for commencing the long sea voyage, shorter trips being advantageously taken in the earlier part of the season.

On the high seas the climatic features of comparative equability of temperature, of considerable moisture, of high and fluctuating atmospheric pressure, are observed together with the freedom from organic and inorganic dust, and richness in ozone, which are also noticeable features of elevated climates. In excessive moisture, in high and variable atmospheric pressure, and in equability of temperature, the sea-climate contrasts with that of the mountain valley. It is well pointed out by Hermann Weber, however, that the diaphanous air of mountain elevations is but little warmed by the rays of the sun, which strike so warmly upon more absorbent intercepting objects, so that the variability of shade temperatures is not so great as it would seem to be. Cases of threatened phthisis, especially of the acquired kind, in those with overworked nervous systems; quiescent disease, especially of the catarrhal type, with defective sanquification, and cases of lymphatic and scrofulous type, do well on a voyage. Hemorrhagic cases, those of more purely tubercular type and those complicated with dyspepsia or diarrhœa, on the other hand, are not adapted for sea voyages.

The sea voyage is unquestionably a most successful means of climatic treatment in suitable cases, and it is instructive to note, that although the conditions are so very different from those of elevated climates, yet, allowances being made for individual peculiarities as furnishing a margin in favor of either, the same classes of cases do well at both.

October is the best month to start on the long voyage to Australasia, and a sailing vessel should be preferred.

In taking a sea voyage to Australia a principal object is, besides the advantages of the voyage itself, to escape the home winter. The voyage out to Melbourne takes some seventy or eighty days, and the vessels are in correspondence with ships for other parts in Australia, Tasmania, and New Zealand. For those who start later, in December, the Orient steamers, which take the Suez route and arrive in forty days, may be preferred.

Unless a sea voyage can be taken with first-class accommodation and with every possible luxury, it should not be advised; moreover, it is important that some definite plan be agreed upon beforehand as to route and destination. A certain steadiness of purpose, in this respect, is essential for those who would profit by their temporary expatriation. Patients who go to the antipodes in search of health and find it on their way, frequently by the time of their arrival have forgotten their original purpose; and scamper about over very various climates at unseasonable times, in a manner calculated entirely to thwart the object with which their journey was designed.

Patients should only remain at Melbourne the briefest possible time, the climate there being most unsuitable for chest diseases; nor is that of Sydney very suitable at the time of year calculated for arrival. Hobart is easy of access from Melbourne either by steamer direct, or to Launceston and thence by rail.

The climate of Hobart is perfect from November to May, and the hotels, boarding-houses, and apartments of the neighborhood are very good. Children do well there, and a patient who desired to remain at the antipodes during our worst months, with the view of getting home again about June, could not do better than spend the interim at Hobart. Australia itself does not at the present time afford any great attractions for chest invalids, except for the more robust and independent, with little or no actual disease, who, invigorated by the voyage out, desire simply to lead a more out-door life than is possible in their own country. For such, the neighborhood of Sandhurst, north of the Victorian range of mountains, the Riverina and suburbs of Sydney in New South Wales, or Warwick and the Downs in Queensland, afford ample fields for pastoral life.

It is often of the highest importance for a promising case to spend at least two winters away, and the patient can for this purpose remain at Hobart until May, and then visit Sydney or the interior of Victoria, Queensland, or New South Wales; or an out-door sheep-farming life may be led in Northern or Western Victoria, except for the two hottest months of December and January, which should be spent in Tasmania or New Zealand. The climate of New Zealand may be said, generally speaking, to be an improvement, for chest invalids, upon Southern England, having more sunshine and warmth and greater possibilities of out-door life, but with a stormy and decidedly variable climate. The choicest parts of New Zealand are perhaps Napier in Hawkes Bay, N. Island, and Nelson in Tasmania Bay, S. Island, *i.e.*, they are warm, fairly equable, and afford good accommodation. The interior of Otago is spoken well of, but it is colder than the other places named.

A shorter voyage may be taken to the Cape, spending a month or two at Graham's Town. Cape Town itself is far too dusty a place to be suitable for chest invalids, and pause may be made on the return journey at Madeira.

Another plan of combining a shorter voyage, with residence away from England for twelve months, would be by P. and O. steamer, so timed as to reach Calcutta after Christmas, spending January and February there, and then in twenty-four hours' journey getting to Darjeeling, where the rest of the year, from March to November, might be passed, returning, after making a further stay at Calcutta, by the Riviera gradually homewards.

By one or other of the plans just suggested, the advantage gained by a long sea voyage may be still further improved, one or two winters being escaped, and the patient returning home at the beginning of the second or third summer. In other cases, after a year or two devoted to recovery, some locality may be fixed upon for permanent residence.

Winter.—(December, January and February). For the objects mainly in view in the climatic treatment of threatened and quiescent early stage cases of phthisis, *viz.*, to secure the enjoyment of air and exercise out of doors, there are still English places fairly well adapted to meet the requirements of those who cannot get abroad. And in cases in which only a period of three months can be spent abroad, it is far preferable to select the three spring than the three winter months. With the setting in of the winter frosts there is no longer the same malarious influence to be dreaded in inland leafy districts, as in the late autumn. Those patients who have homes with sunny exposure and fairly protected from cold winds, situated on dry, porous soils in our inland counties, may do very well, taking exercise between 10 a.m. and 3.30 p.m., and carefully looking to the warming and proper ventilation of their dwellings.

Hastings, Ventnor, Bournemouth, Teignmouth, Torquay, and Tenby (south-side), may be selected by those not well situated at home. Of these places the air of Teignmouth, Torquay and Tenby is softer than at the others, and more suitable for cases with dry, harsh skins and irritable mucous membranes. It is to be remembered that whereas at Hastings, St. Leonards and Ventnor, the coast residence should be chosen, and the uplands only in special instances, at Torquay, on the other hand, the quay residences are cold, damp and misty fsom defective sun exposure and imperfect circulation of air, whilst the terraces on the slopes are comparatively warm, sunny and dry. St. Mary Church, a suburb of Torquay, suits some people better, being more withdrawn from the sea.

Bournemouth is a sea climate, much modified by the extensive moorlands behind it and the pine growths amidst which many of the best houses are placed. The habitations are more isolated and stand farther back from the sea than at other places, and these facts render the climate a happy mixture of marine and moorland, suitable for many people with whom a more purely sea air disagrees. An asthmatic or bronchial element would suggest Bournemouth in preference to other places. In point of warmth Bournemouth is not to be preferred to any of the other south-coast places, but there, as in most of the large places, *e.g.*, Torquay and Hastings, there are many sheltered nooks well known to resident practitioners; indeed these places may be said to be "full of climates!"

The Riviera climates have in common the advantages of warmth, brilliancy, accessibility, and excellence of accommodation, and one or other place amongst them is generally selected for those for whom the elevated resorts are not desirable or are unsuited.

Early catarrhal and quiescent pneumonic cases do best here, as in the hill resorts; but where the mountain climates agree, and the patients are enabled to remain there for six months or longer, the results are often of a more permanent kind. Cases of great anæmia will do best to spend the first winter at all events in the Rivera. In many cases, also, in which the necessity for change of climate occurs when cold weather has already set in, it is more prudent to select one of the warmer resorts with the view perhaps of getting on high ground in good time before the ensuing winter.

There are many early stage cases of phthisis of the tubercular type, complicated in some instances with threatened or actual laryngeal or bowel troubles, in which the prognosis is bad; and yet provided the patient can go abroad with every comfort and accompanied by his family or nearest friends, relief of symptoms and lessened activity of progress may be thus effected.

Children and young adults, especially girls who have had acute bronchial or pulmonary affections, with a delicate family history, may, by one or two winters spent in the Riviera, completely and permanently recover. In elderly people again, with phthisis, in whose tissues senile decay has already commenced, the warmer climates of the plains are admirably adapted.

Hyères.—The west-end of Hyères is the best part, its elevation permitting of superior drainage. Somewhat removed from the sea this part of Hyéres enjoys a slightly modified sea climate, and partially protected from winds by the chain of islands parallel to, and two or three miles distant from the shore. The climate is more still and soothing than many of the Riviera stations; excitable people of the "erethric" type will probably do better there than elsewhere. Some forms of asthma, bronchitis, and emphysema, also do well. From November to February the climate is good, in February and March the mistral is sometimes severely felt. During the period stated, cases of advanced but quiescent phthisis, and those complicated by albuminuria, may do well here.

Cannes.—For prolonged residence Cannes affords the most varied attractions, perhaps, of any of the Riviera stations. The upland parts, away from the shore, should be selected for residence. The climate is decidedly bracing and brilliant, but somewhat changeable. Cases of scrofulous and lymphatic habit, with lax secreting membranes, nonpyrexial sweatings, cases of anæmia, senile forms of bronchitis, emphysema, asthma, and persons, both young and old, recovering from acute chest disease, do well here. On the other hand, persons of highly nervous temperament, with a disposition to sleeplessness, do not do well.

Of more advanced cases, the later hectic period of pneumonic phthisis with cavity formed, limited in extent but still secreting, often do very well here. In this large district there are many climates, and it is safest to take local advice as to any precise locality for prolonged residence.

Cimiez, a suburb of Nice, although, not as yet, very well provided with accommodation, is well suited for asthma cases, neuralgic people, and bad sleepers. It is less liable to variable winds than the town of Nice. For elderly people Nice has the advantage of level walks and promenades and town life, with the brilliant Riviera sunshine.

Monte Carlo and Les Moulins, in the eastern bay of the principality of Monaco, are amongst the choicest spots of the Riviera, for residence; the dwellings being between two and three hundred feet above the sea level, well protected from mistral by mountains behind, yet with abundant air circulation. These places are adapted, however, only for wealthy people in limited numbers.

Turbie-sur-Mer.—"Admirably situated in a small rock-bound bay just a little to the west of the jutting promontory on which the castle and old town of Monaco are built. It is protected on all sides except towards the sea, and from its own natural advantages, as well as from its nearness both to Nice and Monaco, it is surely destined, when properly developed, to be a popular resort."[1] This embryo resort is situated at an elevation of 2,000 feet.

Mentone.—This resort, especially the East Bay, is more sheltered than the other stations, and, whilst warmer than they are, has a less free circulation of air. It is well adapted for short periods of residence in cases of sub-acute bronchial catarrh, gouty bronchitis, and phthisis with albuminuria. It, is, however, not suitable for the hemorrhagic forms of phthisis. Cases of somewhat advanced phthisis find this locality soothing, and persons who lead, from cardiac diseases or other causes, lives of enforced inactivity, will often profit by sojourn here.

San Remo is a resort not easily to be distinguished from Cannes. The air is somewhat more moist. As a rule the same cases do well as at Cannes. There are many other places of resort along the northern coast of the Mediterranean, Malaga, Costabelle, Bordighera, Alassio, Ospedaletti, Pegli, Genoa, Spezia, Pisa, and others, all of which have their merits and fair accommodation.

On the way to the Riviera, Arcachon, Biarritz and Pau, may be mentioned. *Arcachon*, although colder than the more southern stations, is well adapted for many cases of arrested phthisis, of rather excitable than depressed nervous system. The winter villas amongst the pines are alone to be selected, and riding exercise can be enjoyed amongst the interminable pine forests. Some cases of chronic phthisis with excessive secretion do well here. The climate is also well adapted for asthma. Considerable shelter from the spring winds is obtained amongst the pines.

Of *Biarritz* I have already spoken as a highly marine climate, suitable for the late autumn and early winter months, and for early stage or threatened cases of phthisis. People with a disposition to neuralgia are said not to do well here.

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[1] Burney Yeo. Health Resorts, p. 407.

Pau, an inland climate more suited for threatened than declared disease. Cases of chronic, bronchial, and laryngeal catarrh, and some old patients who do not flourish at the Riviera climates, are better here. It is a sedative climate with magnificent surroundings and sheltered walks and drives.

Algiers. A somewhat modified sea climate at the port Mustapha Supérieur, about two miles from the cliff, which is most suitable for invalids. An equable, dry and warm climate, but little affected by mistral, and rarely visited during the winter and spring months by the dusty south wind.

Cairo is for the most part unsuited for chest invalids, the air being very dry and dusty. Daheebiah excursions up the Nile may be suggested as an interesting change for the wealthy, and adapted for cases of early quiescent phthisis.

Madeira is a warm, moist, equable climate well adapted for the bronchitic forms of phthisis, especially those in which the disease supervenes upon old-standing winter cough. Jaccoud includes Madeira, Algiers, Palermo, and Pisa, in the same group, as peculiarly suitable to the chronic stage of pneumonic phthisis; and the point in these climates which he regards as of the greatest importance, viz., meteorological uniformity, combined with an effect which is tonic rather than debilitating, he finds more especially to appertain to the two former places, and *par excellence* to Madeira.[1]

At different elevations from Funchal, up to a height of 2,000 ft., residences can be obtained, so that owing to the slight difference between the mean temperature throughout the year "the rule enjoining prolonged residence, which is never more imperious than in pneumonic phthisis, can be preserved without the least difficulty."[2] The absence of dust (and of mosquitoes) is especially emphasized as a feature of this climate.

California.—My experience of the Pacific health resorts, Santa Barbara, Los Angeles, etc., is too limited to do more than allude to them as affording winter quarters adapted for those with disease too advanced for the high American climates, or in which those climates are for other reasons undesirable.

Spring.—(March, April, May.) Of localities suitable for chest invalids during spring, our islands can boast of but very few. There are certainly numerous nooks and corners with sunny exposure and protected from cold winds, where on porous soil and at a moderate elevation residences exist or might be built, singly or in small groups, very suitable for invalids who are unwilling or unable to go further. At some of our seaside places, Bournemouth, Torquay, St. Mary Church, Grange, Isle of Wight, sheltered spots can be found. Bridge of Allan, by Stirling, N.B., is one of the best spring places with which I am acquainted. Here on the southern slope of the hill, protected from the North and East, are houses, and a considerable hydropathic establishment with sheltered walks.

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[1] The Curability and Treatment of Pulmonary Phthisis, p. 371.

[2] Loc. cit., p. 372.

Throughout the continent the same difficulty presents itself, more or less, with regard to the avoidance of irritating cold spring winds. With the exception of this occasional drawback, most felt during February and March, the whole Riviera is at its best during the spring season.

Mentone (E. Bay), Monte Carlo, Trubie, and Ospedaletti, neither of which two latter places is as yet developed, are said to be most protected but it is perhaps scarcely wise, on account of occasional cold winds, to shift quarters that are otherwise suitable.

Grasse, 1000 feet, a bracing hill suburb of Cannes, is a good late spring resort. Les Avants above Montreux is also protected. Many patients who have spent the winter at the Riviera, are tempted at this season to move on to Florence (where, however, the spring winds are much felt) or Rome.

Madeira and Algiers are not affected by severe spring winds, but the latter place becomes too warm for residence in the later spring. Arcachon, Biarritz, and Pau, are fairly good spring climates; all, however, possess but imperfect shelter.

In May, Montreux or Méran may be visited with the object of commencing the grape cure.

Summer.—(June, July, August.) In the summer months the travelled and tired invalid will generally do best to return to his home and famliair haunts, friends, and diet. June is a favorable month for visiting certain health resorts and baths, as Ems, Aix-les-Bains, Aix-la-Chapelle, Allevard-les-Bains, Royat, Mont Dore, Eaux Bonnes, but chiefly for the treatment of special throat, or other symptoms, to be incidentally alluded to elsewhere.

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CHAPTER XXXI.

TREATMENT OF PHTHISIS—(*Continued*).

ACUTE FIRST STAGE CASES—ACUTE SOFTENING AND FORMATION OF CAVITIES—SUMMARY.

ACUTE FIRST STAGE.—When there are signs of recent consolidation with pyrexia, rapid pulse, troublesome cough and expectoration, perhaps blood-stained, the change of air treatment, strongly to be advocated for prodromal catarrh or incipient or threatened phthisis, frequently proves disastrous. Such patients require complete rest, careful nursing and treatment, until the acute period of their illness subsides.

Rest in the recumbent posture at an equable temperature of about 62° is of the first importance. Milk, light puddings, nourishing broths and cooked fruits should be freely allowed. Stimulants as a rule avoided. The secretions must be cleared, and in cases where the circulation is much quickened and the skin dry and hot, an effervescing saline mixture, containing three to five minims of tincture of aconite, may be given in the course of every four hours, for some eight or twelve doses, after which the aconite should be omitted. Presently small doses of iodide of sodium or potassium in an alkaline mixture should be substituted, and to it may be added, when the temperature has abated, the hypophosphite of soda, to be taken three times in the twenty-four hours. When pain is complained of, a small area below the clavicle may be painted with epispastic solution, or in very acute cases a couple of leeches applied.

The night-perspirations at this early period of the disease are rarely of a severe character, and it is wise as a rule not to check them by any specific treatment. If the temperature at night range high, it may be moderated by tepid sponging and quinine, and half a grain of opium may be given if the cough causes restlessness. Five grains each of pulvis antimonialis comp. and Dover's powder will sometimes prove a happy combination. Under this kind of treatment the fever will commonly subside in a few days, either wholly or to a moderate evening rise of temperature to 99° or 100°.

When this period has been attained, the patient may be allowed to move about a little, and in fine sunny weather to get out into the fresh air. Plans may be made for removal to a suitable sea-side or inland place, preferably to a pine-growing or gravel soil, and at a fair elevation. But the accommodation and care in diet and cooking to be met with, must largely determine the locality chosen.

It has been suggested in cases of one-sided disease in the earlier stages to ensure rest to the diseased portion of the lung by certain mechanical appliances. "Lung splints" have been designed by Dr. Dobell for this purpose, and Dr. McCrea,[1] of Belfast, has recommended that the chest should be strapped.

I have tried in a few cases at the Brompton Hospital the effect of restraining the movements of the chest in apex catarrhal-pneumonia by keeping the arm on the affected side in a sling. It is difficult to say that any distinct advantage has been derived from this treatment. The naturally very slight expansile movements of the apices become still further restricted in disease without mechanical aid, and the object of rest to the part is sufficiently secured by insisting on that freedom from effort or exercise, which shall secure the most complete functional rest possible to the respiratory and circulatory systems during the first fibrile period of the disease.

In the inflammatory basic complications which are very apt to supervene in the course of phthisis, especially when the pleura is involved, I have very frequently obtained good results by restraining the chest movements by the application of a broad piece of strapping of sufficient length to extend round the semi-circumference of the chest, and for an inch or two beyond the median line in front and behind. I have not found it necessary in such cases to adopt the more thorough method of strapping the chest, recommended by my colleague, Dr. Fred. Roberts,[2] in the treatment of pleurisy. Immediate relief to pain, and amelioration of the local signs and general symptoms, usually follow this treatment. If necessary, local anodynes or counter-irritation may be combined with mechanical rest, by including beneath the strapping a belladonna plaster or piece of spongio-piline sprinkled with dilute iodine or some such application.

In the early stages of catarrhal pneumonia now under consideration, troublesome cough is sometimes a symptom calling for treatment: when attended with scanty and difficult expectoration, and, if the cough be of a violent, straining character, the addition of a few minims of opiate to the mixture will be useful. It is often better practice to have recourse to a light respirator charged with five or six minims of oil of eucalyptus or of pine, and one or two minims of oil of bitter almonds, dissolved in ten minims of chloric ether, to be worn for five or ten minutes to half an hour at intervals of three or four hours during the day, and to give a single half grain dose of opium or codeia at bed-time. It will often be observed, as in the case related at page 199, that the cough remains troublesome, while the pulmonary signs are greatly improving and all secretion sounds rapidly drying up. Dr. Thorowgood[1] has drawn attention to the irritable dry cough which is so frequently attendant upon the subsidence of pulmonary disease, and truly observes that the patients should be encouraged to check the cough themselves as much as possible. This they can do to a great extent, and may be assisted by some sedative cough mixture, if necessary, to secure rest at night. The morning cough in these cases—and, indeed, in many others—is the most troublesome. It is, however, the natural consequence of a good night's rest, and should never be checked by a sedative, since the matters suitable only for expectoration, if retained considerably, impede respiration, become highly irritating and set up further inflammatory and specific troubles in the lungs. A cup of warm cocoa or tea or milk taken before rising will greatly facilitate expectoration. If this does not suffice a simple steam inhalation is useful, or a small dose of ether and ammonia. The old-fashioned remedy of rum and milk taken early in the morning, is useful for this purpose; a dessert-spoonful of rum to a claret glass of warm milk being sufficient.

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[1] Lancet, Sol. ii., 1874, p. 76.

[2] Practitioner, vol. xii., 1874.

On the subsidence of fever, or in more serious cases this abatement to an evening rise of temperature only, cod-liver oil may be given, and some tonics of arsenic, iron, mineral acid or alkaline bitter, as the general and particular features of the case may suggest.

The uses and administration of cod-liver oil in phthisis.—The advocacy of the use of cod-liver oil in phthisis by Dr. C. J. B. Williams and the late Dr. Hughes Bennett, has gained for this remedy a recognized value second to none other in the treatment of the disease. Cod-liver oil is regarded by most authors rather as a food than a medicine. Its easy assimilation and absorption by virtue of the biliary constituents with which it is inpregnated, render it a valuable nutrient: it in some way appears to quicken cell development,[2] and to arrest degenerative processes in a manner that cannot be accounted for by the quantity taken: one to four drachms, two or three times a day, being the average dose. In the administration of cod-liver oil the object is to commence as soon as possible, and to continue for long periods rather than in large doses. One teaspoonful may be taken twice a day as soon as solid food can be digested (Williams), and if well-borne the dose may be gradually increased to a maximum of one tablespoonful three times a day, beyond which dose few patients can go with advantage. M. Jaccoud, however, insists upon the necessity of taking heroic doses, commencing with one tablespoonful daily, after a week two tablespoonfuls twice a day, and similarly increasing up to six or eight tablespoonfuls or even larger doses daily. An examination of the stools will discover the passage of oil by the bowel if too large doses are taken.

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[1] The Climatic Treatment of Consumption.

[2] C. J. B. and C. T. Williams, Pulmonary Consumption, 1871, p. 343. Jaccoud agrees with Williams in the opinion that the oil has a resolving effect upon the local lesions of phthisis

The best time for taking oil is about one hour after meals, when the stomach is occupied by food in a condition prepared for escape through the pylorus, for it is beyond the pylorus that the oil becomes absorbed; but some patients will in practice be found better able to assimilate the remedy at other times. The pale and comparatively tasteless oils are as a rule better taken and digested than the brown oils, and for the past twenty years but little of any other than the purest pale oil has been dispensed at the Brompton Hospital.

Many people can take oil out of the bottle, others take it in milk or on orange wine. Fresh lemon-juice is a favorite, and in certain cases a very valuable vehicle for the oil. Cold coffee with a pinch of salt sprinkled on the surface of the oil is sometimes preferred. In children and sometimes in adults, Parrish's food or steel wine are excellent vehicles, and various other agreeably flavored tonics may be combined with oil, *e.g.*, quinine wine with a little hypophosphite of soda or lime, or phosphoric acid with strychnia and infusion of orange. Some patients prefer Cognac or a liqueur mixed with the oil, and M. Jaccoud suggests one-third or one-fourth as much liqueur as there is oil for each dose.

Many persons who have no difficulty in taking oil complain much of eructations afterwards. This objection is sometimes insuperable, and from the discomfort and anorexia caused, renders a continuance of the oil impossible. In many cases it is due to the oil not being taken at the proper time after food, in others it is due to an undue acidity of the stomach requiring correction. I have known many cases in which the addition of a little pancreatine powder in the proportion of one grain to each drachm, placed on the surface of the oil with or without a little salt, has completely prevented eructations. In other cases Messrs. Savory & Moore's pancreatine wine may be used as a vehicle. Some patients can best take the oil in a single dose at bed-time.

Dr. Williams recommends that patients taking oil should avoid pastry, fat meat, rich stuffing, and partake only sparingly of butter, cream and sweet things.

Certain additions may be sometimes with advantage made to cod-liver oil; thus, a drop of creosote or $\frac{1}{100}$ grain of strychnia (Williams) are sometimes valuable correctives. The addition of fifteen or twenty minims of ether is supposed to increase the digestibility of the oil by stimulating pancreatic secretion; in some few cases it will be found useful. In a few cases it will be also found that the addition of an alkali by saponifying the oil, will enable it to be better borne and absorbed.[1]

Unquestionably the most favorable periods for giving oil are the apyrexial intervals of phthisis, and M. Jaccoud is very emphatic in the opinion that only at these periods can the drug be assimilated. In smaller doses, however, the remedy can sometimes be taken with advantage, if not during the comparatively brief continued fever of phthisis, at least during the more prolonged hectic of the disease.

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[1] Brompton formula.—Misi. Olei Morrhuæ Preparata. ℞ Olei Morrhuæ ʒvj., Liq. Ammoniæ fort. ♏ij., Olei Cassiæ ♏j., Syrupi ʒij. Dosis ʒij.

Substitutes for cod-liver oil.—M. Jaccoud maintains that when from pyrexia, disordered stomach, or high atmospheric temperature, oil cannot be borne, *glycerine* may be taken easily and with great advantage. M. Jaccoud gives the glycerine in doses of one tablespoonful three times a day, with the addition of one drop of essence of mint, and two drachms of brandy or rum.[1]

In larger doses this hydro-carbon may give rise to symptoms of loquacity, agitation, and insomnia (Jaccoud), analgous to those of alcoholism; a degree or two of elevation of temperature may also be occasioned.

I cannot say that I have been able to observe any marked benefit to result from the administration of glycerine, nothing in the least degree comparable to the effects of cod-liver oil.

In certain cases where oil cannot be taken, *pancreatic emulsion* is unquestionably of service. It should be given in dessert-spoonful doses, carefully stirred in milk warmed to a natural temperature. Sometimes a liqueur glass of black coffee or some alcoholic liqueur, may be added to the mixture thus prepared.[2] The best time to take the emulsion is about two hours after food.

Creams and *fats* may be similarly prescribed. A piece of mutton suet allowed slowly to dissolve in a tumbler of milk warmed by standing on the hob or in a slow oven, is an old-fashioned remedy for phthisis; the milk thus treated should be filtered through muslin before being taken. Preparations of *malt* are useful in some cases for those who cannot as yet digest oil, and there are also elegant preparations of malt with cod-liver oil, cream, hypophosphites, etc., which may be tried on occasions.

Olive oil taken alone, or in the form of salads or with sardines, is an imperfect substitute for cod-liver oil.

Of general tonic remedies in the early periods of phthisis and in the debility prodromal to the disease, arsenic is one of the most valuable. The arseniate of iron or small doses of arsenious acid may be given in pilules with food twice a day. Jaccoud recommends gr. $\frac{1}{10}$ of arsenious acid to be first taken at the commencement of two principal meals, increasing the dose each week by two pilules daily, until six, eight or ten are taken daily; this treatment being continued for two or three months unless signs of intolerance of the drug are shown. After this period the dose should gradually be diminished. This remedy is especially valuable in cases in which iron is not well-borne.

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[1] Op. cit., p. 143.

[2] To be added to the milky mixture, not to the emulsion before the milk.

If under the arsenical treatment the bowels become relaxed, the tongue coated, digestion painful, or appetite fails, it must be at once restricted or withdrawn.

Unless specially indicated, strychnia and quinine are of no particular service at this period. The hypophosphites are of undoubted value, and iron in small doses and for short courses is often very valuable, the iodide being chosen in scrofulous cases. This period of phthisis, the activity of the disease having subsided, is the most suitable for treatment by climatic change, and I have in a previous chapter pointed out, as far as possible, the kind of change best adapted to meet particular cases, temperaments, and seasons.

ACUTE PNEUMONIC PHTHISIS.

Period of acute softening and formation of cavities.—In the treatment of the active periods of the more caseous pneumonic forms of phthisis, the particular symptoms to be regarded are:—1. Pyrexia. 2. Nervous prostration and bodily exhaustion. 3. Certain symptoms especially observed at this stage, *viz.*, wasting, anorexia, night-sweats, cough and expectoration, hæmoptysis, intercurrent pleurisies.

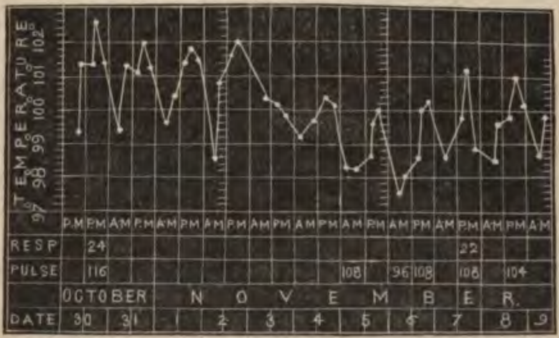

FIG. 23.

Pyrexia.—It can scarcely be doubted that the fever at this period of phthisis is mainly dependent upon some form of purulent absorption. The often very decided effects of remedies point to the same conclusion, for they are remedies—quinine, arsenic, salicylic acid—of an antiseptic kind. The employment of inhalations at this period is also largely governed by this view.

The hectic type of the fever prevalent at this period will be sufficiently recalled to mind by a glance at the subjoined chart; it marks the suppurative process which is active in bringing about the removal of diseased structures.

Quinine in from three to five-grain doses dissolved in milk and taken between meal times, is often of value in controlling hectic and in sustaining the patient. A tablespoonful of whisky may often with advantage be added.

Arsenic cannot be given in these cases in sufficient doses to diminish the temperature in any striking degree, but it has a marked influence upon some of the most distressing symptoms attendant upon the fever, and it seems sometimes in a remarkable manner to improve the general condition of the patient. This drug is most indicated in those cases in which daily recurring chills are complained of. Three to five drops of the liq. arsenicalis or the arseniate of soda solution, or $\frac{1}{12}$ to $\frac{1}{4}$ of a grain of the arseniate of iron three times a day, will often suffice to entirely prevent the recurrence of these chills. Arsenic has also been extensively used for this purpose by my colleague, Dr. Pollock, and generally with good result.

Salicylic acid is a drug which has a marked effect upon the temperature of phthisis, and especially at the period under consideration. In the case whose temperature is depicted in the foregoing diagram, the drug was commenced on the morning of the 3rd of November, and the immediate fall of temperature is well shewn. The chart, however, also shows what will be frequently found, viz., a return of pyrexia after a few days' use of the drug, necessitating a larger dose. In cases, however, of less extent of lesion, the fall of temperature has proved enduring. Another effect of the salicylate, to which I drew attention in the former edition of my work, is the manner in which it appears to diminish wasting and to preserve the weight and strength of the patient, even although the fever be not perfectly controlled. The cough is often favorably influenced by this drug, and the expectoration somewhat lessened. The salicylates may be detected in the sputum, and may thus possibly have some local influence on the cavities.

On the whole I have obtained better results from this drug as regards the treatment of pyrexia at this period than from any other. It should be given in ten to twenty-grain doses in an aromatic and slightly bitter mixture every three or four hours, or in larger doses once or twice a day at periods calculated to anticipate the diurnal rise of temperature.

It is quite obvious, however, that in many of the cases now in view, there is to be taken into account, not only the softening down of caseous products and suppurative processes in the walls of recently formed cavities, the symptoms of which may be referred to purulent infection; but also fresh consolidation and caseation proceeding, over which it is doubtful whether, save in an indirect way, antiseptic drugs have any influence. And in many cases the suppurative manufacture of the pyrexia is so active as to be only very partially under our control, so that when any particular remedy is vaunted as a specific for the fever of this period of phthisis, successful in all cases, its repute is based upon imperfect observations, or very limited experience.

Amongst the unhappy effects of salicylic acid, must be named its physiological action, which is attended with noises of bells in the ears, deafness, and sometimes great depression of spirits. The temperature is apt to become depressed below the normal, so that it is often desirable only to give the drug during certain periods of the day.

Antipyrin, a most valuable drug for reducing temperature, especially in enteric fever, does not appear to have the antiseptic properties of salicylic acid, and is less adapted for cases of prolonged high temperature. When the daily rise of temperature is excessive, the drug may be given in ten or fifteen-grain doses for the two or three hours preceding the expected rise. Its effects upon the patient must be carefully watched, howver, and if it be found to cause any marked nervous depression, or if it merely serves to postpone the rise of temperature to another period of the day, it had better not be persevered with. Excessive sweating is frequently caused by antipyrin; this may be checked by $\frac{1}{100}$ grain of atropine, or gr. $\frac{1}{20}$ agaricin, given about half an hour before the first dose of the daily series.[1] Vomiting is occasionally caused by this remedy, and its occurrence is a sufficient reason for omitting. In cases where the stomach is too sensitive to tolerate antipyretic remedies, rendering it desirable to give as few medicines as possible, the evening temperature can be moderated by the employment of tepid sponging with a solution of dilute acetic acid 1 part, water at a temperature of about 85°, 6 parts, and Eau de Cologne $\frac{1}{4}$ part. As an adjuvant to other remedies, especially when there are night sweats, this application is also of value.

Iodoform, *Creosote*, and *Tar* have been recommended for internal administration on account of their antiseptic properties, during this hectic stage. My own observation leads me strongly to doubt whether they can be given in sufficient quantities to influence pyrexia without running grave risk of setting up gastro-intestinal irritation, and destroying appetite. They have much less diffusive power than the remedies already spoken of, and are of more value in other apyrexial conditions under which their use will be again considered. When there is much local disturbance of stomach and upper bowel, however, small doses of creosote in combination with opium are sometimes of great service; it is most important in regard to this point to remember, however, the highly irritating effects of the products of caseous softening and cavity secretion when swallowed, upon the stomach. Many people, delicate-minded females and sensitive men, feel a delicacy or a timidness in expelling the products of their disease. Children often do not know how to do so. This inability or unwillingness to eject the sputa is a fruitful source of stomach and bowel troubles, and possibly of local tuberculosis; a simple warning will often prevent the necessity for special medication.

Topical medication of the lungs.—For the period of phthisis now under consideration, moist inhalations in the form of medicated vapors, are as a rule better avoided. Except for cases in which throat symptoms are prominent, sprays are also of no value, since it is very doubtful whether they penetrate beyond the larynx or main bronchi. There is no doubt, however, that by the inhalation of the dry vapors of volatile antiseptics, much advantage may be gained in this stage of phthisis.

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[1] Yearbook of Treatment, 1884, pp. 21 and 306.

The effect of such inhalations is to relieve cough and to lessen expectoration, and there can be no doubt that, as I believe to be the opinion of Dr. Coghill, who has so ably advocated their use, one of their chief functions is *to do away with cough mixtures.*[1] For certainly the cough linctus treatment of this eliminative period of phthisis by lulling cough and deranging stomach is the very worst that could be devised. Our great objects are to get rid of the effete products of caseous liquefaction and suppuration, and to keep the pus-secreting surface as disinfected as we can do without harming the patient in the process.

As has been well pointed out by Dr. Solomon Smith,[2] the cavity contents are on the one hand in contact with living tissue, which has a retarding influence upon bacterial activity; thus our antiseptic measures receive aid from within, and if whilst using antiseptic inhalations we by hygienic and tonic treatment increase vital resistance, we may hope to "turn the balance in favor of the healing process, and against the active germination of the bacilli."[3]

Of the innumerable respirators invented, Duncan Mackenzie's, and Blake's, are I believe the best for thoroughly introducing the inhalant, but the majority of patients find such close respirators very irksome, and can be persuaded to wear the more open and lighter patterns devised by Burney Yeo, and others, for a longer time and with less fatigue. I myself more generally prescribe the oral respirators of Coghill, Robert, and Wordsworth. The latter is a very light and comparatively elegant respirator, consisting of two curved and perforated plates of vulcanite, so fitting as to enclose a layer of cotton wool. It is the best for use with eucalyptus or pine oil, but for carbolic acid and creosote the metal respirators of Coghill and Roberts are better adapted and less apt to blister the patient.

Dr. Coghill's formula[4] is a favorite and much used one, *viz*:—"Tinct. iodi, ætherealis, acidi carbolici, āā ʒ ij., creosoti vel thymol, ʒ j., spiri. vini. rect. ad ℥ j., M. Where the cough is urgent or breathing embarrassed, chloroform or sulphuric ether may be added at discretion."

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[1] Antiseptic Inhalation in Pulmonary Affections, by J. Sinclair Coghill, M. D., British Medical Journal, vol. i., 1881, p. 841.

[2] British Medical Journal, vol. i., 1884, p. 353.

[3] Year book of Treatment, 1884, reference by the author to Dr. Smith's views, page 19.

[4] Loc. cit.

I find three drms. eucalyptol or ol. pini sylvestris to the ounce of rectified spirit, or spirit of chloroform, a good combination: ten to fifteen drops on the wool of an oral or naso-oral respirator, or twenty drops for the more open Yeo's respirator, to be worn for half an hour to an hour or longer after the first morning expectoration, in the middle day, and in the evening. If the cough be troublesome, one drm. of oil of bitter almonds may be added to the 1 oz. solution, and double strength spirit of chloroform used as the solvent. I have lately, I believe with advantage in some cases, used a simpler method of dry inhalation, *viz.*, directing the patient to quilt a layer of Lairitz pine wool into a piece of flannel some six or eight inches square, and sprinkle upon it a few drops of the pine oil, then roll it up to form a tube and breathe through it for a few minutes together frequently through the day, keeping a piece of the wool saturated with the oil suspended in the bed-room.

Austin's inhalation tube has been much recommended; its advantage is its portability and the frequency with which it can be used without attracting notice. I have recently had under observation an intelligent Frenchman, with a large cavity on one side, who averred that he could feel the antiseptic penetrate more deeply when inhaled through the nostril by this means than by the use of respirators, with which he was familiar. Dr. Ward Cousins has also designed a small tube for oral inhalation. Both the latter inhalers are better adapted for the medication of the less active and more chronic cavities than for those now under consideration.

Dr. Hassall has pointed out the small amount of antiseptics evaporated during the use of respirators, his remarks especially applying to carbolic acid and creosote, and he recommends the use of chambers charged with the substances required by evaporation, from large surfaces aided by warmth. Such chambers would be cumbersome and difficult to arrange in private houses, and in hospitals could only be used by a few, and it is doubtful whether the patients would not suffer more from the deficient supply of air than they would gain by the lung medication.

After the use of the inhaler at bed-time, a sedative may be given to allay cough and give rest.

Night-sweating is a symptom usually calling for treatment from time to time during the eliminative period of phthisis. It arises from two principal causes, *viz.*, fever and nervous exhaustion, these causes being commonly combined, but the one or the other predominating in different cases.

There are innumerable empiric remedies for night sweats, all of them successful in certain cases: they should not be had recourse to, however, until such rational measures have been adopted as are indicated by the causative conditions above alluded to.

The patient must be steadily supported by nourishment during the day, and some readily digestible food given the last thing at night; fever must be moderated by tepid acid sponging at bed-time, and a third dose of the quinine or bark and acid, or arsenical tonic that may be in use, given a little before bed-time. If these means prove insufficient, then aid may be sought from specific remedies.

In cases in which the night-sweats are occasioned by reaction from fever, and deluge the patient on waking, towards the morning hours, when the temperature begins rapidly to fall below the normal, an extra dose of quinine will often serve to check them. Extract of belladonna gr. $\frac{1}{2}$, or atropine gr. $\frac{1}{80}$–$\frac{1}{50}$, may be given in combination with quinine or alone. When the cough is troublesome, hyoscyamine gr. $\frac{1}{100}$ to gr. $\frac{1}{80}$ may be preferred. Arsenic in the form of the arseniate of iron pill $\frac{1}{8}$ to $\frac{1}{4}$ gr. will prove useful in cases in which nervous prostration is marked. In other cases, especially of the latter kind, when the hectic has lasted some time and has resulted in much nervous exhaustion, strychnia is useful in full doses at bed-time, but this remedy is sometimes unfortunate in causing wakefulness. Four-grain doses of oxide of zinc or two grains of valerianate of zinc will sometimes answer alone or in combination with belladonna.

Picrotoxin gr. $\frac{1}{80}$, agaricin gr. $\frac{1}{20}$, eserine gr. $\frac{1}{60}$, physostigma (extract $\frac{1}{10}$ gr. in pilule three times in the night,) are other remedies of occasional value.

It is of the utmost importance to have some easily assimilable food, such as good cold beef-tea, or beef essence ready to hand, should the patient wake up, and especially with night perspiration, for the sweats are profoundly exhausting from the large quantity of saline material discharged, and the nervous exhaustion thus induced tends to perpetuate their nightly recurrence. The immediate supply of a stimulating salt-containing food tends thus to remedy the loss and to prevent its recurrence. Patients liable to night-sweating should wear a thin flannel loosely fitting over-gown, and a fresh night-dress should always be ready-aired for changing.

General summary.—Throughout the variable but often prolonged period of suppurative fever through which the patient has to pass during the softening and elimination of the caseous products in pneumonic phthisis, the objects of the practitioner must be:

(*a*) Steadily to support the patient by an abundant and well-assorted dietary, rich in fats, in salts, in nitrogenous elements. In most cases a fair amount of alcohol in the form best adapted to the peculiarities of the case is required.

(*b*) The hygienic surroundings must be strickly looked to (p. 280), and as much fresh air as is compatible with the season of the year and the patient's strength, allowed.

(*c*) Particular symptoms, pyrexia, dyspepsia, sweatings, cough and expectoration, will suggest the line of medicinal treatment, which must be determined upon after careful consideration of the whole case, and not lightly changed. A written sketch of the dietary and times of taking medicine will be a useful guide to friends and nurses, and a wholesome check against mixtures "every four hours," and cough linctus "occasionally," besides pills for night-sweats, and local applications, which wholesale medication if long endured cannot fail to be disastrous.

(*d*) No climatic change of any radical kind is indicated for this period of phthisis. As a rule the treatment is best carried out at the patient's own home. For the poorer classes our large special hospitals are admirably adapted. In cases where the home is situated in the midst of damp and unhealthy surroundings, a removal to a high and dry locality or to the sea-side, with sunny exposure, cannot fail *cæteris paribus* to be beneficial.

CHAPTER XXXII.

QUIESCENT, SECRETING AND ULCEROUS CAVITIES—FIBROID STAGE —TUBERCULAR PHTHISIS—BRONCHI-ECTATIC, DIABETIC, DUST, AND SYPHILITIC PHTHISIS.

QUIESCENT, SECRETING AND ULCEROUS CAVITIES.—After the active period of phthisis during which one or more cavities have been forming, although the pulmonary disease may have ceased to extend, there still remain lesions which require attention. In a certain number of cases the cavities, varying in size and number, become quiescent, and slowly contract, yielding less and less secretion. In these cases of drying and contracting cavity, the cough becomes irritable and the patient frequently complains of its "tightness," having been accustomed to easy expectoration whilst the secretion was abundant. He should be encouraged by explaining to him the favorable nature of the cause of his difficulty, and directed to check so far as possible by an effort of the will the tendency to violent cough. Sedative cough mixtures, injurious whilst the expectoration was abundant, now judiciously timed, become of much value. More or less morning expectoration persists for a considerable time after the cough has ceased during the day, and should not be checked by sedatives. The further medicinal and climatic treatment of cases of this kind must be conducted on general principles.

It is of great importance to keep such patients in a pure atmosphere, since they are most sensitive to all septic conditions. Under any unfavorable hygienic circumstances the cavities become secreting, and other surrounding centres of disease take on fresh activity.

Moorland, sea and mountain air are all suitable for these cases, and one or other should be advised in accordance with indications special to the individual case, where abundant air space and facilities adapted to the strength of the patient in the way of riding, driving, bath-chair exercise, level walks, can be obtained.

As the patient's strength improves, the development of the sound portions of the lungs may be encouraged by carefully regulated exercise on rising ground. It is in this latter phase, provided the *extent* of lesion be not great, that these cases are amongst the most suitable for the mountain resorts.

Secreting cavities may, as already pointed out, persist from the active stage or be developed by renewed activity of the lining of cavities, which had become quiescent.

The objects we have in view in the treatment of such cases are:—1. To lessen secretion. 2. To promote its evacuation; and 3. To disinfect such cavities.

Counter-irritation is useful over the cavities in the form of strong iodine applications, or flying blisters, or perhaps a blister kept open for a time by savin ointment. Acids and astringent iron tonics with cod-liver oil are needed. Tar in the form of the syrup of the U. S. P., or Guyot's capsules, also creosote or eucalyptus internally, are sometimes useful; but for the reason already alluded to, viz., that these remedies in order to be efficacious under the conditions present must be given in full doses, and are then liable to disorder the stomach; it is as a rule better to rely upon counter-irritation and topical medication by inhalations with general tonic treatment. Sedative cough mixtures are directly contra-indicated save at bed-time, and then solely for the purpose of procuring rest.

It is in these cases that inhalations are most useful, for—firstly, there being no actively spreading disease present, they are not contra-indicated: secondly, we can, by their use, render less nocuous the pus that bathes the surfaces of the cavity and which is apt to become inhaled into distant parts of the lung: thirdly, there can be little doubt that appropriate inhalations sometimes have a healing or alterative effect upon the internal surface of the cavity. Dry inhalations by means of respirators are alone of service. Coghill's formula of carbolic acid and iodine, also the pine oil and eucalyptus inhalations, are very useful. The respirator charged with the material for inhalation should be worn for an hour in the morning after the first expectoration, again in the afternoon and for two hours in the evening, and the air of the bed-room may usefully be kept fragrant by pine-wool suspended, sprinkled with some oil of pine.

Sea-side resorts, the more bracing of the Riviera stations and Algiers, are the climates most suitable for these cases.

Ulcerous cavities.—Cases of ulcerous or active cavities are always to be regarded with suspicion, as probably due to insanitary surroundings, and such evil conditions must be first looked to and remedied. These cases are of an erysipelatous type, and are best treated by quinine in full doses, or perhaps tincture of iron, and locally by sedative inhalations of carbolic acid, conium, and chloroform with hot-water vapor,[1] or eucalyptus oil[2] with hot water. Ipecacuanha wine used as a spray with a steam atomizer[3] is sometimes useful.

After the more active general symptoms have lessened, if the blood-stained, and copious expectoration lead us still to infer that the walls of the cavity are hyperæmic, if not ulcerated, the best treatment will be found to be free counter-irritation. A blister should be applied over the site of the cavity, and should be kept freely discharging for a week or ten days by means of savin ointment dressing. I have seen the active local symptoms completely subside under this treatment, and the expectoration, from being abundant and sanguineous, become scanty and viscid, apparently consisting of bronchial mucus only.

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[1] Chloroform. ♏x., Succi conii, ʒj., Glycerini acidi carbol., ʒij., Aq. Bullientis, ℥viij. Brompton Hospital Ph.

[2] Eucalypt. ol., ʒiij., Mag. carb. lævis, ʒj., Aq. distillatæ, ℥iij., ʒij. in aq. calore (150°) pro inhal.

[3] Codman and Shurtleff's, and Siégle's, are the best.

I have, as yet, said nothing about the local treatment of phthisical cavities and consolidations save by vapor or spray inhalations. But the treatment of both these conditions, by puncture and injections through the thoracic wall, has been attempted by several physicians. Nor do I think that their efforts, albeit in some instances ill-timed, merit the hasty and indiscriminate censure of the late Dr. Hughes Bennett,[1] who concludes a sweeping condemnation of all measures of local treatment whether by inhalations or otherwise, with the following remark:—"The result of all these efforts has been—what an intelligent consideration of the pathology of the disease might have anticipated—a uniform failure."

Dr. F. Mosler of Griefswald[2] relates three cases of cavities in the upper part of the lung, two of which he injected with solution of permanganate of potash through a fine Dieulafoy's syringe. In the third case he introduced a "thick silver drainage tube," which he covered with carbolized lint and through which much purulent secretion escaped. In this case, on the subsequent occurrence of hæmoptysis, he injected through the tube a weak solution of perchloride of iron. It does not appear that in these cases the patients were injured by the treatment. Dr. Wm. Pepper of Philadelphia[3] has also treated three cases, and in one case with, he believes, positive benefit. His plan was to employ local anæsthesia, and the patient having taken a full breath, to introduce a Dieulafoy's No. 1 (finest) needle. He then injected four to ten minims of diluted Logol's solution (liquor iodi. B.P.) in the proportion of four min. to ʒ i water. Dr. Pepper suggests that "superficial circumscribed indurations or caseous infiltrations of the lung tissue may have introduced into them through fine needles such injections as may tend to induce absorption or reparative action."

The important part attributed to bacilli in producing the lesions of phthisis, has helped to revive the treatment of tubercular consolidation and localized cavities by injections, and Professor Lepine and M. Truc of the Faculté de Lyon, have recently employed an alcoholic solution of creosote, two to four per cent. of which they have injected from a few drops to twenty cubic centimetres in fifteen cases. The general result has been negative, however; no material harm, and no notable improvement having been affected.[1]

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[1] Reynolds' System of Medicine, vol. iii., p. 589.

[2] Ueber lokale Behandlung von Lungenkavernen von Dr. Fr. Mosler, Prof. in Greifswald.—Berliner Klinische Wochenschrift, Jahrg. 10, 1873, p. 509.

[3] Philadelphia Medical Times, March, 1874.

CHRONIC AND FIBROID STAGES OF PHTHISIS.—The management of the chronic and fibroid stages of pneumonic phthisis consists chiefly in the prevention of fresh catarrhs by judicious clothing, the selection of climate when practicable, the avoidance of night-air, and protection from irritating fogs, or cold or damp winds by respirators, together with a nutritious but not stimulating diet. Iodine frictions, soothing or antiseptic inhalations (carbolic acid being particularly useful when there is any fœtor of expectoration), seem the best local remedies; whilst the general condition, including that of digestion, the nature of the cough and amount of expectoration, supply us with indications for the administration of appropriate drugs—iron, cod-liver oil, strychnia, alkalies, tonics, etc.,—or warrant the withdrawal of all medicines.

There is only one special remark concerning the treatment of these chronic indurative cases of phthisis, during the often-extended period of quiescence, which seems called for, and it is this—that though such cases require careful *surveillance*, and for several years, where practicable, carefully selected climates to suit the different seasons of the year, they do not require the persistent administration of tonic medicines and cod-liver oil. They improve immensely under such remedies up to a certain point, which may be readily recognized by the medical attendant, and cannot be better described than by saying that it amounts to the most perfect health attainable by a patient who has had a certain area of respiratory surface cut off. If beyond this point we persevere with iron and oil and too nourishing or stimulating a diet, we may still further increase weight and heighten color, but the pulse quickens, the patient gets more short of breath; he becomes in a word plethoric and liable to pulmonary congestion and hæmoptysis, or to dyspepsia and diarrhœa. A rapid neutralization of all the good results obtained, with great danger of fresh and perhaps fatal renewal of the old disease, is thus the consequence of too great anxiety to again arrive at a degree of health and bodily vigor which is impossible with a permanently damaged lung.

TUBERCULAR PHTHISIS.—Acute and chronic tubercular phthisis must be treated on the same general plan as the pneumonic forms, but with much less hope of, in any material degree, staying the progress of the disease. This progress, although often less palpable to auscultation, is nevertheless more real and unrelenting.

Cases of acute tubercular phthisis should not be sent away from home, and herein lies the importance of an accurate diagnosis. In doubtful cases it is far better to put off a definite opinion as to the desirability of change for a short time, and to watch meanwhile the effects of rest and treatment upon the progress of the case. In the presence of a sustainedly raised temperature, no move from home should be advised.

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[1] Essai sur la Chirurgie du Poumon, par H. Truc, M. D., Paris, 1885, pp. 151-159; also Lyon Medical, 1885, t. xlix., p. 8.

In *Chronic Tubercular Phthisis* again no distant change of climate should be entertained. A sea voyage would be ill-advised. The patients as a rule have irritable digestions and nervous systems, with harsh, dry skin, and quick feeble pulse, often also laryngeal or bowel complications, or threatening of such; and dry, exciting or elevated climates only aggravate their symptoms. Arcachon, Pau, Mentone, Hyères are, of continental places, the best adapted to these cases at suitable seasons, and provided they can go comfortably and with responsible friends, the effects of a warm somewhat sedative climate are soothing and palliative in the winter and spring. Of home places, Torquay, Ventnor, Tenby, Hastings, are preferable for the autumn months and early summer.

BRONCHI-ECTATIC PHTHISIS.—In this form of the disease the profuse secretion is sometimes manifestly controlled by the administration of tar in the form of the Pil. Picis of the Pharmacopœia or Guyot's capsules, or other preparations. The texture of the unaffected lung is rarely sound, so that elevated resorts can but seldom with safety be advised. The dry climate and open-air life of the Orange States in early stage cases may be tried.

The most rational treatment consists in directing the patient twice a day to place himself in such a position—leaning down with the head low, and a slight inclination away from the affected side—as shall lead to the most complete emptying of such dilated tubes and bronchi-ectatic cavities as may be situated towards the base of the lung, and immediately afterwards to inhale some antiseptic vapor, *e.g.*, eucalyptol or oil of pine, by means of Robson's dry spray apparatus,[1] and to be out in fresh, dry air as much as possible.

The strength must be well maintained by a liberal dietary and moderate amount of stimulants. The question of operative treatment in these cases has been already touched upon (pp. 154, 162).

DIABETIC PHTHISIS.—This variety calls for no special remark in treatment. In such cases a considerable abatement in the régime for diabetes is usually necessary, and codeia, quinine, and cod-liver oil are indicated.

DUST PHTHISIS.— A complete and timely removal of the patient from the cause of the disease, and the pursuance in future of an out-door pursuit in a healthy country or seaside locality may result in complete recovery.

SYPHILITIC PHTHISIS.—Syphilitic disease of the lung and phthisis having its origin in syphilitic lesions must be treated with anti-syphilitic remedies, and very brilliant results will be obtained in many cases. Mercury may be given in the form of the perchloride alone, or in combination with iodide of potassium, the dose of each being graduated in accordance with the strength and susceptibility of the patient. A course of mercurial inunction is sometimes of great service, and such patients may be sent to Aix-la-Chapelle with a plea in mitigation, however, of the somewhat too heroic treatment they may there meet with.

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[1] This apparatus consists essentially of a bellows, to be worked by the foot, sending a current of air through tubes filled with pumice saturated with the oil, and escaping through a perforated nozzle, shaped like the rose of a watering can.

When the hectic phenomena of phthisis are in combination with syphilitic manifestations, Donovan's solution (liq. hydrarg. et arsenici hydriod.) may be given with advantage.

Cases in which phthisis has supervened upon a constitution previously undermined by syphilis must be carefully discriminated, and specific remedies used in them with caution.

CHAPTER XXXIII.

ULCERATION OF THE BOWEL—LARYNGEAL PHTHISIS—HÆMOPTYSIS —VOMITING WITH COUGH—INTER-CURRENT PLEURISIES, ETC.

ULCERATION OF THE BOWEL IN PHTHISIS.—In the treatment of ulceration of the bowel in phthisis, it may be usefully remembered that the mere presence of such ulcers does not necessarily cause diarrhœa, this symptom, when present, being due to attendant catarrhal inflammation of the adjacent mucous membrane, or to increased peristalsis set up by an active condition of the ulcer. We have thus three conditions to bear in mind in the treatment of tubercular ulceration of the bowels, viz:—(1) Acute ulceration—what may be called the first stage of the disease, in which the ulcers are in process of formation or activity. (2) Chronic ulceration, with diarrhœa alternating with constipation. (3) Chronic ulceration, with intercurrent diarrhœa arising from errors of diet.

Acute ulceration.—In this condition the diet must be carefully regulated, so as to give as little residue as possible for the bowel to digest. On the diagnosis being established, the patient must be kept warm in bed, and the diet restricted to milk and a little animal broth, in the form of good beef-tea or meat essence. About four ounces of milk or beef-tea should be allowed every two hours in such portions that twice as much milk as beef-tea be taken. The milk may be given warm or cold in accordance with the desire of the patient; occasionally a little crushed ice may be added to the milk, at other times it may be boiled with rice and strained, when six ounces may be allowed instead of four. Sometimes koumiss in the third degree of fermentation may be substituted for milk, and when the stomach is very irritable, peptonized foods may be given. The beef-tea may be varied with veal-tea, chicken or mutton-broth. After a few days Savory & Moore's malted oatmeal may be prepared with the milk twice a day, or a little white of egg diffused in it. Rusks and a little tea may be allowed, and the diet slowly improved through such grades as custards, ground rice puddings, invalid turtle, scalded bread and milk, scraped underdone or raw meat, grated chicken, etc. As the diet is improved the intervals between the times of taking food may be increased to three or four hours.

In some cases a small quantity of stimulant in the form of pale brandy, added to each second quantity of the milk, is of service, or a little old port wine may be allowed.

During the acute periods now under consideration, and whilst any tenderness remains, the patient will be best in bed, and a hot linseed poultice should be kept applied over the abdomen. When there is marked tenderness over the region of the cæcum, a blister three inches square may with advantage be there applied under the poultice.

In medicinal treatment it is of much importance, first of all to clear away any irritating matter from the bowel, and the administration of from ʒ j. to ʒ ij. of castor oil well shaken up with a couple of ounces of hot milk, and a dessert-spoonful of Cognac added, will prove of great service. It is often a good plan to give a very small dose, gr. ½ to gr. j. of calomel, two hours previous to the dose of oil. After the lapse of some few hours, to allow of the effects of these medicines to take place, ten or fifteen grain doses of bismuth with gr. ½ of ipecacuanha, and ten grains of soda in mucilage and chloroform water is the best routine treatment: after some eight or twelve doses, a little chalk mixture should, if needed, be added in place of the soda. Sometimes cerium will answer better than bismuth, and in cases of painful peristalsis small doses of opium are needed.

It is the greatest possible mistake to treat tubercular diarrhœa with astringents and opiates as a matter of routine, and without very carefully looking to the diet. This treatment is on a par with that of pulmonary lesions by sedative cough mixtures, and only serves to mask symptoms whilst the tubercular lesions are spreading and deepening.

In chronic ulceration, if diarrhœa supervene upon previous constipation, or if the appearance of the motions and coated tongue, lead to the inference that sybalous or irritating materials are present in the bowels, a dose of castor oil or compound rhubarb powder should first be given, the dietary carefully gone over, although with less strictness than in the acute stage, and a course of bismuth and soda or chalk prescribed.

In severe cases twenty grains of bismuth and five grains of Dover's powder is a useful combination, or ten grains of each of bismuth and compound kino powder may be given every three or four hours. In a much more limited number of cases, sulphuric acid and opium answer better. A layer of pine wool quilted on flannel should be kept firmly applied to the abdomen. On the subsidence of active symptoms a little calumba, cascarilla or quinine may be given twice a day in combination with fluid bismuth, or five-grain doses of hypophosphite of lime may be prescribed with ten minims of liq. calcis saccharati in calumba or chiretta infusion.

Constipation is very apt to follow upon diarrhœa in cases of ulcerative disease, and a collection of solid fæces tends to re-awaken the activity of the ulcers. The smallest available dose of laxative should therefore be prescribed when necessary, ʒ j. of castor oil being frequently sufficient when taken well emulsified in hot milk. The diet may be modified, and a little fruit, cider or light beer allowed to correct the tendency to constipation. In cases where the third stage koumiss has been taken during the period of diarrhœa, a return to medium koumiss may be suggested as less astringent.

The above plan of treatment will be found most generally efficacious in cases of moderate intensity of acute and of chronic intestinal ulceration, and during periods of intercurrent diarrhœa.

In advanced cases, however, our therapeutical resources are often taxed to the utmost, and in vain, to effect more than a passing relief. A remedy which scarcely ever fails to give temporary relief, even in the worst cases, is the starch and opium enema of the Pharmacopœia. Some acetate of lead may be added to this enema, or the lead and opium suppository may be employed with advantage. In more chronic cases and those of greater severity we still have a large armory of more decided astringents to fall back upon, amongst which acetate of lead and sulphate of copper (gr. ¼) with opium hold a high place. But the vegetable astringents, kino, catechu, hæmatoxylin, tannic acid, Indian bael, may be each in turn tried in combination with opium with decided, but often only temporary, benefit. The aromatic chalk and opium powder of the Pharmacopœia given in a mixture containing tincture of catechu is a favorite remedy in these cases. Although all the vegetable astringents owe their efficacy to the tannin they contain, yet there is some peculiarity in each, and when one has failed another will often succeed, again in its turn to lose its virtues in the particular case. Almost all these cases require opium in addition to the astringent, and sometimes opium alone given in the solid form is the best remedy.

The diarrhœa, however, in all advanced cases should when possible be restrained by careful diet, with the help if necessary of a suppository at night.

Laryngeal Phthisis.—The treatment of the laryngeal form or complication of phthisis, although in many cases confessedly unsatisfactory, is yet generally attended at least with relief.

We have, in all cases, two conditions to treat, laryngitis and laryngeal ulceration: the latter, having once occurred being the abiding lesion, the former (laryngitis) being more or less intense at different times and sometimes subsiding altogether. As in the lungs and in the intestines, so in the larynx, it is the attendant inflammation that causes the chief symptoms and calls especially for treatment. Now and then the local application of a strong solution of nitrate of silver (twenty grs. to the oz.) or of sulphate of copper (twenty grs. to the oz.) is useful. The former especially sometimes lessens sensibility of the parts. But I am quite sure that in this disease too great a diligence in the use of local applications to the larynx is to be deprecated. I have not found much benefit result from the use of astringent solutions in the form of sprays. But such sedative or slightly stimulating inhalations as hyoscyamus, tincture of benzoin, hops, etc., are often very useful, and I have found great advantage from the employment of the ipecacuanha spray recommended by Drs. Ringer and Murrell for bronchitis. When there is much pain in the larynx, the persistent use of external counter-irritation will be often found most beneficial. A small blister the size of a shilling should be daily applied over the region of the larynx for several days, so as to keep up constant, but not too severe, counter-irritation. This treatment may be combined with sedative inhalations, and rarely fails to give considerable relief. Where the difficulty and pain in swallowing are great, the application of a ten-per-cent. solution of cocaine may be made to the throat, or gelatine lozenges containing five-per-cent. of the drug allowed slowly to dissolve half an hour before meals. In some cases nutrient enemata should supplement, or for a time wholly replace, the ordinary method of feeding; they give rest to the diseased parts and lessen the terrible sense of exhaustion from which the patients often suffer.

The dyspnœa in these cases never becomes urgent enough to suggest the expediency of tracheotomy which on other grounds would not be thought of.

When ulceration has ensued, the insufflation of iodoform or boracic acid 1 part, cocaine or morphia ⅛ part and pulv. tragacanth 1 part, by means of a quill or insufflator once or twice a day, will render the ulcers less active and painful. Dr. Semon[1] recommends iodoform, acid borac. āā gr. j., morph. acet. gr. ⅙. Respirators charged with eucalyptus and bitter almond oil are often of considerable service, but in some cases cannot be worn.

In extensive ulcerative destruction of the cords, the most distressing symptoms arise from the strain of cough and the mechanical difficulty of expectorating. These truly terrible symptoms are most difficult to relieve, and when they arise, a few drops of chloroform may be inhaled, or 5 minims of iodide of ethyl; finally recourse may be had to morphia to lessen the acuteness of the distress.

Apthous mouth and throat, a very distressing complication, especially apt to occur in diarrhœa cases, may be warded off for a long time by careful cleansing of the mouth each time food is taken, with weak boracic lotion, or with tepid water just made pink with toilet Condy, with a few drops of Eau de Cologne added.

HÆMOPTYSIS.—In speaking of the treatment of hæmoptysis, I have in mind the causes of hemorrhage which I have already indicated as operative in phthisis, viz.:—Active hyperæmia, diseased small vessels, and erosion or aneurismal dilatation of large vessels. The two former conditions accounting for most attacks of primary hæmoptysis and such intercurrent attacks as are preceded by, or attended with, the symptoms and signs of fresh accession of lung disease. The latter condition being answerable for the severe and sometimes fatal hemorrhage proceeding from cavities most commonly in the later stages of the disease.

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[1] St. Thomas' Hospital Reports, vol. xii., 1883, also Yearbook of Treatment, 1884, p. 290.

Primary Hæmoptysis.—Shock is a marked symptom in early hæmoptysis, especially in first attacks. The agitation of the patient must be calmed by the confident assurance of the absence of immediate danger, and all measures of treatment should be carried out without bustling about or whispering. The doctor will often treat his patient best by quieting anxious friends. The recumbent posture with head and shoulders slightly raised by pillows, should be adopted with light coverings and warmth to the feet. Ice should be given, and all stimulating restoratives strictly forbidden.

In all cases of hæmoptysis the aspect of the patient, and character of pulse should be noted, the temperature taken, and the stethoscope lightly applied over the front of the chest without allowing the patient to breathe with more than ordinary depth: the heart's sounds should also be carefully listened to. All these observations can be made without disturbing the patient in the least, and information as to the attack can be gained from friends.

There are different types of cases to be observed for which different plans of treatment are indicated. *Firstly*, in a considerable proportion of cases there are pallor, chilliness, pinched features, and small, quick, thready pulse of combined nervous shock, loss of blood, and constitutional feebleness; the first outburst of hemorrhage will have passed. If hemorrhage be still going on, 20 gr. of gallic acid with glycerine and camphor water may be given, followed by 5 gr. doses in iced water every half-hour for three or four doses, then at longer intervals.

If the hemorrhage be severe, a full dose of liquid extract of ergot may be given, ʒ ij. to ʒ iij in iced water and, ♏xx. to ♏xxx. every hour afterwards for a few doses. In other cases turpentine[1] answers better: this drug is, however, best held in reserve, as a rule, for use in case the other remedies fail. Acetate of lead is a favorite remedy with some authors; it is a drug, however, of low diffusion power, slow to reach the part where its action is wanted, and difficult to eliminate when its effect has been accomplished; moreover, its primary action of constringing bowel secretions is directly opposed to the requirements of the case. As a general rule, cases of primary hæmoptysis subside without much trouble, and any remedy used in a sufficient number of cases, will gain a reputation. Salt and water was long held by the Brompton nurses as of great efficacy. As soon as active bleeding has ceased I am content to give a little dilute sulphuric or nitro-hydrochloric acid, and ipecacuanha.

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[1] Olei terebinthinæ, ♏xx., Mucilaginis acaciæ, ʒ iij., Aquam cinnamom. ad ℥ j., Brompton Pharmacopœia.

Tincture of hamamelis, in ten to twenty minim doses, I have found to have useful influence in checking the passive hemorrhage that sometimes succeeds to an active outburst.

There is much diversity of opinion as to the desirability of giving opiates in early hæmoptysis. Inasmuch as I regard the secondary consequences of blood retained in the lungs as possibly harmful, I am myself unwilling to give opiates to check the cough, unless this be very rending and violent. In cases, however, in which the shock and terror of the patient are very marked, an opiate (gr. ½ to 1 opium, and gr. 1 camphor) may be of great service in calming agitation and quieting heart's action. I have never been tempted to adopt Trousseau's plan of giving an emetic to arrest hæmoptysis. It is of great importance to relieve blood pressure in hæmoptysis, and a saline purge should be given at an early opportunity in the attack.

For the hæmoptysis which sometimes occurs in persons with venous plethora, usually of intemperate habits, and which not uncommonly ushers in a rapid inflammatory form of phthisis, the best treatment is not by direct astringents; a drachm of sulphate of magnesia with ♏15 each of tincture of digitalis and aromatic sulphuric acid, taken every four hours until the secretions become free and watery, is far more efficacious, and in some cases it is well to begin with a mercurial. In very exceptional cases of this kind, depletion from the arm may be advised. I have seen two instances of smart hæmoptysis in pregnant women with contracted mitral valve, in whom the precedent evidence—full venous system, laboring right ventricle, and strongly thudding pulmonary second sound—might have suggested a timely venesection, and saved a dangerous illness.

In cases in which there is distinct evidence of the syphilitic cachexia, full doses of iodide of potassium should be given either alone or in combination with ergot. In these cases the hæmoptysis is sometimes very severe. After a primary hæmoptysis, when the shock of the hemorrhage has passed, an observation of the temperature range will give most information as to the nature of the case, and prompt future treatment.

Recurrent hæmoptysis.—In the treatment of this form of hæmoptysis, which it will be remembered (page 243) is due to the rupture of a vessel or aneurism in a cavity, besides the general principles of absolute muscular rest, etc., before referred to, we must be more diligent with astringents and remedies which control the heart's action and allay cough: *ergot*, acting upon the muscular walls of the arteries, *digitalis*, diminishing the frequency of the heart's action, and *opium*, lessening excitement and allaying cough, are of the greatest value. Our object is to allow the blood to coagulate at the seat of rupture, and faintness short of actual syncope should be encouraged, rather than prevented by stimulants. Nauseant remedies, however, from their relaxing effects on the vessels, are inadmissible. Interrupted cold applications to the chest may be tried in these cases more usefully, I think, than in those in which the hemorrhage is capillary.

The tendency to recurrence of hemorrhage in these cases must be remembered, and care must be taken not to allow the patients to get about too soon. In those patients, too, who are gifted with rapid blood-making powers, and who pick up flesh with great rapidity after hæmoptysis, a judicious abstinence from butchers' meat, and the complete withdrawal of stimulants, may ward off or postpone the next attack.

The hemorrhage in this form of hæmoptysis is far more profuse than in primary hæmoptysis, and is often immediately fatal. In some cases it recurs again and again, until the patient arrives at the lowest ebb compatible with life, when the bleeding vessel is staunched by coagulum, and recovery slowly commences.

With reference to prophylactic treatment, patients the subjects of phthisis, particularly with chronic cavities, should be cautioned against muscular efforts, such as hurrying upstairs or walking fast, catching trains, etc. The experiments of Colin[1] show that on exertion the pressure of blood in the pulmonary artery increases in greater ratio than that in the aorta..

Intercurrent Hæmoptysis.—In the course of phthisis hæmoptysis may at any time occur, being due to active hyperæmia of a fresh portion of lung, or rupture of a vessel in a portion already diseased. The treatment will be in accordance with the nature of the case. Slight staining of the expectoration, or even a small quantity of fresh blood, not infrequently appears in course of the elimination periods of the pneumonic forms of phthisis; such occurrences need occasion no modification of treatment, being due to the breaking across of tubercular vessels of small size and already partially occluded. As a measure of precaution stricter quietude must be enjoined for a short time after any such occurrence. Mixed sanguineous expectoration in cavity cases is best combated by free counter-irritation by the application of a blister, and subsequently dressing the surface with savin ointment for a week or ten days.

Spurious hæmoptysis.—I have dealt with the treatment of this non-pulmonary form of hemorrhage, in the chapter devoted to its description (see p. 245).

VOMITING WITH COUGH.—This distressing symptom is especially characteristic of the more indurative forms and stages of the disease. Its successful treatment is attended with much difficulty. The symptom is primarily due to the mechanical difficulty in expelling secretions from cavities and bronchial tubes which are surrounded by dense, tough, airless consolidations; and, secondly, it it is distinctly to be observed in many cases that there is undue irritability of the vagus, giving rise to cough directly food is taken into the stomach. Any catarrhal condition of the stomach must be treated by alkalies, bismuth and hydrocyanic acid taken an hour before meals. If there appears to be an hyperæsthetic condition of the vagus, a little hydrocyanic acid with ℥10 doses of liq. morphiæ hydrochl., taken half an hour before the principal meals, either alone or with bismuth and soda, may succeed in preventing the cough. In other cases again, a course of strychnia as a nerve tonic is very valuable, and pepsin may be given after meals. The patient should keep very strictly at rest for an hour or so after meals.

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[1] Comptes Rendus, p. 759, 1864.

INTER-CURRENT PLEURISY AND PNEUMONIA AND BRONCHITIS.—Inter-current pleurisy at the upper half of the chest, is best treated by the application of small blisters or liq. epispasticus. If the pain be rather of a dull, aching, than of a sharp, pleuritic character, and if physical signs point to the presence of fresh catarrhal pneumonia or local bronchitis, the more gradual effect of iodine applications is to be preferred. The liq. iodi., B.P., may be painted over the part each day or second day for two or more applications, a layer of pine-wool covered with oiled silk being kept constantly applied over the part painted. Another good way of applying iodine is by the use of iodized cotton-wool, a French preparation in the first instance, but Messrs. Savory & Moore have recently prepared an iodized wool of two definite strengths, *viz.*, twenty per cent, and ten per cent. A layer of No. 1 (twenty per cent. strength) may be first applied for twenty-four hours covered with impermeable tissue; by the end of that time, or perhaps after thirty-six hours, the color will have been discharged, and a decided counter-irritation effected which may be kept up by the application of No. 2 (ten per cent.), and its renewal each second or third day. It is best to apply the fresh wool in the morning so that the pungency of it may not interfere with sleep. It must also be remembered that some skins are much more sensitive to iodine than others, so that the weaker preparation is strong enough in some cases.

When some part of the lower half of the chest is affected with pleurisy in the course of phthisis, the prompt application of a long piece of Leslie's strapping three or four inches broad, so as to extend round the affected side to a couple of inches beyond the median line in front and behind, will by restraining respiratory movements at once relieve pain, and often arrest the local inflammation. This treatment is of great value in many cases, and is to be particularly advised in those cases in which, in addition to the signs of pleurisy, superficial crepitation with a few liquid clicks suggest that liquefaction of caseous pneumonic centres is proceeding close under the pleura. The timely application of strapping in such cases, will, by relieving the lung from the shock of cough, often avert pneumothorax. Some patients cannot bear this application, however, and the rough clinical test of its probable usefulness in any given case, is to hold the side with steady pressure by the hand, and see if relief from pain is thereby obtained. In cases where strapping is not tolerated, the application of a small blister with a hot linseed poultice over it, will speedily give relief. When the pain is severe, a blister may sometimes be applied with great advantage under the strapping.

TUBERCULAR MENINGITIS.—The early stages of tubercular meningitis are, as already pointed out, very insidious and obscure, and it must be confessed that the progress of this fatal malady is beyond our control. Some of its more distressing symptoms may, however, be mitigated. A brisk calomel purge should first be given, and if pain in the head be a marked symptom, it will be relieved by the application of cold lotions or the ice cap. In severe cases, marked relief will be afforded by the application of leeches to the temples. The room should, in the earlier stages of the disease, be darkened; care must be taken to relieve the bladder if necessary.

Full doses of bromide and smaller doses of iodide of potassium, seem sometimes to give relief. When the twitchings are marked, chloral should be given in combination with the bromide. Should convulsions threaten, the chloral should be sufficiently pushed to avert if possible this symptom, so distressing to friends although happily unfelt by the patient. The chloral may be administered in a little water or barley-water, by the rectum, if it cannot be taken by the mouth. A few whiffs of chloroform has sometimes appeared to restrain convulsions.

CHAPTER XXXIV.

ON ABSCESS AND GROWTHS IN THE MEDIASTINUM.

ABSCESS in the mediastinum is a somewhat rare disease and is probably never of primary origin. The causes of abscess in this situation may be thus enumerated.

1. Injury to the sternum.
2. Post-sternal syphilitic node.
3. Perforation or injury to the œsophagus from the impaction or penetration of foreign bodies.
4. Septic causes; pyæmia, enteric fever, tracheotomy.
5. Glandular suppuration in association with scrofulous or tubercular disease: sometimes occurring after whooping cough.
6. Caries of the spine.

SYMPTOMATOLOGY.—A history of a blow or of some other circumstance of the kind alluded to in the above list of causes, and the discovery of symptoms and signs pointing to mediastinal inflammation, are the elements of diagnosis in this often obscure disease. *Pain* and *tenderness*, *pressure signs*, only very moderate in degree, and *pyrexia*, are the symptoms most worthy of notice. The pain is seated behind the sternum or between the shoulders and radiates from these points. Paroxysmal cough of the laryngeal type, a certain amount of obstruction to venous return and some pain or difficulty in swallowing, are the pressure phenomena which may be observed, chiefly in cases of abscess of considerable size in the anterior mediastinum. The pyrexia assumes the hectic type and is attended with rigors and sweatings.

On inspection, a certain degree of fullness may be noticed over the superior sternal region, and sometimes there is also a red blush over the surface and a slight œdema masking the outlines of the cartilages and spaces. There may be some obscure impulse communicated from the aorta, and, in cases of large collection of pus behind the sternum, this impulse may closely simulate that of aneurism. In posterior abscess there is usually prominence and tenderness of one or two of the dorsal spinous processes.

The percussion and auscultation signs of posterior mediastinal abscess are very obscure, so that the frequent association of spinal caries, with its characteristic external signs, is of much importance in diagnosis.

In abscess of the anterior mediastinum, there is dullness over the region of pain and swelling, with unduly conducted treacheal breath-sound. In cases in which the aortic sounds and impulse are conducted, it is important to make several examinations at times when the patient is at rest and free from cardiac excitement.

If let alone the abscess may point externally or into the pleura, pericardium, lung, or bronchus. In posterior mediastinal cases, the abscess may burrow along the vessels between the pillars of the diaphragm and finally point in the iliac or femoral region. Most commonly anterior mediastinal abscesses, either point externally or burst into a bronchus.

TREATMENT.—In the earlier stages, poultices should be employed, especially in those cases in which there is any external tenderness. In cases connected with caries of the spine the appropriate treatment for that affection must be adopted. Complete rest is in all cases necessary. The patient requires to be well supported by general treatment, and quinine administered for the control of hectic.

When the abscess is within reach of surgical treatment it should be evacuated. In more doubtful cases the aspirator may be employed. Cases, however, in which there has been a communication effected with a bronchus, should not be interfered with hastily, as the abscess may become completely evacuated and cured in this way.

MEDIASTINAL GROWTHS.

PATHOLOGY AND ETIOLOGY.—Growths in the mediastinum are of two kinds, sarcoma (including lymphoma) and carcinoma.

As a primary affection carcinoma is rare: its occurrence in this situation being usually due to extension from neighboring parts, especially from the œsophagus, more rarely from the stomach, liver, kidney, or lung. As a secondary deposit from distant sources, cancer of the mediastinum is also rare. The form in which true cancer appears in the mediastinum, is usually scirrhus, but both epithelioma and encephaloid are found as secondary growths.

Primary sarcoma of the mediastinum is almost invariably lymphomatous: any of the other kinds, round-celled, spindle-celled, osteoid, or enchondromatous, may occur secondarily in accordance with the nature of the primary tumor.

The bronchial or anterior mediastinal glands are usually the primary seat of lympho-sarcoma: in some cases the disease appears to commence in the remains of the thymus. The growth having once broken through the capsule of glands, tends rapidly to infiltrate the mediastinum, involving the pericardium and creeping along the vessels and bronchi to the root and interior of the lung. In their later stages these growths are exceedingly malignant, small lymphatic tumors being disseminated through many distant organs.

Age.—Sarcomatous growths in the mediastinum may occur at any age, even in early childhood, and they are more prevalent before than after the middle period of life. They are also of more frequent occurrence in females than males: in both these respects differing, it will be observed, from aneurism. True cancer rarely occurs before middle life and is rather more prevalent amongst males.

Whilst, as a rule, the most important symptoms and the fatal issue of the case, are attributable to the mediastinal lesion, it is important to note, that growths limited to the mediastinum are but very rarely met with: one lung, more commonly the left than the right, being generally involved.

SYMPTOMS.—As a rule, excepting of course those cases in which the œsophagus is early obstructed, emaciation is not a marked symptom in mediastinal tumors, but in all cases there has been some loss of weight. The symptoms of mediastinal growths are due to the presence of a foreign solid mass within the thorax, diminishing breathing space, compressing vessels and nerves, and thrusting aside or invading important passages, organs, and tissues. Pain in the chest is sometimes of the darting occasional character peculiar to growths, more generally it is a sense of uneasiness rather than actual suffering, except where the pleura has become involved. The pressure symptoms of growths are insidious in their occurrence, but more persistent and less variable than those of aneurism. Thus the dyspnœa is, as a rule, much less fluctuating than that of aneurism, and it tends more gradually to increase. Paroxysmal dyspnœa is less common, except perhaps in the later stages of the disease. Cough, dry and teasing, or attended with slight and difficult expectoration, is one of the first symptoms complained of. It has a peculiar clanging or husky obstructive character, which the experienced ear at once notices. In the later stages, when the growth has invaded the lung, sanguineous expectoration may be observed, and sometimes a profuse hæmoptysis occurs, which is followed by a considerable, though temporary, amelioration of symptoms. Hæmoptysis is not, however, an important sign of mediastinal growths.

Dysphagia is a much more common and, when present, a more abiding symptom in growths than in aneurism. Pressure upon veins is again, as a marked symptom, of more common occurrence in growths, and the head, neck, and upper extremities sometimes present a turgid œdematous appearance which is very characteristic, large tortuous veins also frequently course over the surface of the epigastrium and chest.

PHYSICAL SIGNS.—A slightly staring, suffused, and anxious expression of countenance is most commonly to be observed, and in marked cases the aspect assumes that of semi-strangulation, pitiable to witness, the swollen and œdematous head, neck, and upper limbs contrasting with the natural appearance of the lower half of the body.

The respirations are distinctly quickened, the pulse generally accelerated, whilst the temperature remains low.

When the anterior mediastinum is involved, as is the case in the majority of instances, some prominence of the upper sternum may be noticed. Enlarged glands at the root of the neck, or in the axilla, should be carefully felt for, the fixed or movable character of which may give a clue to the cancerous or lymphomatous nature of the tumor.

The heart is displaced in a direction varying with the position of the growth. Most commonly it is thrust to one side; sometimes the base is lowered, the apex being tilted upwards and outwards. At other times the growth extends from above downwards between the heart and the sternum, at other times again forwards from behind the organ, and I have met with an instance in which the heart was borne forwards compressed and fluttering against the anterior chest wall.

Over the region of the tumor there is percussion dullness, which encroaches in well-marked cases both upon the mediastinum and lung. The disease is rarely symmetrical, the chief extension of dullness being to one side, but in all cases the middle line is encroached upon. On careful percussion it will be observed that, on the lung side of the growth-extension, the circumference of the area of dullness is bounded by pulmonary note, and this feature is very characteristic of mediastinal tumor as distinguished from primary lung disease: the distinction from pleuritic effusion in which the circumferential parts are first involved, the central last, being still more evident. Some impulse, communicated from the aorta, may often be felt over the dull area, the knocking character of which can usually be distinguished from that of aneurism. The vocal resonance and fremitus are both increased over the area of dullness. The heart's sounds are well conducted, and impulse may be still more distinctly appreciated by the ear. A systolic murmur is occasionally to be heard over some portion of the dull region, wanting, however, in the expansive rough quality that would be expected in a murmur generated by an aneurism of such dimensions.

The respiratory sounds over the tumor are more or less bronchial, sometimes intensely so, at others partially or completely obscured by stridulous rhonchus.

Over the lung on the side to which the tumor most encroaches, and especially over the posterior base, the respiratory murmur may be enfeebled or annulled, and in obscure cases of small deep-seated mediastinal growths, this failure in the entrance of air on one side is, in the absence of signs of effusion, of great importance in diagnosis. Marked stridor on one side would in such cases also be an important sign.

On inspection and also in placing the hands evenly on the two sides over the lower region of the chest, impairment of mobility may be distinctly observed on the affected side during deep inspiration. As the disease advances, in cases in which vital passages are not so immediately involved as to speedily terminate life, the lung becomes more involved from root to periphery, until the whole side is completely dull and distended. Observed now for the first time, it may be impossible to distinguish such a case from one of extensive pleuritic effusion, for the heart is displaced, as it would be by fluid in the pleura, and vocal fremitus, resonance and breath-sound are all suppressed in consequence of the occlusion of the bronchi by growths. The introduction of a fine trochar which fails to produce fluid, and is felt to penetrate and to be fixed by solid tissues, is the only certain means of effecting diagnosis, although careful examination will often reveal out-lying islets of resonance corresponding with thin areas of lung encrusting the periphery of the growth. In other cases, effusion either mechanical from pressure upon the azygos veins, or inflammatory, complicates the signs and symptoms of tumor.

Portions of lung not directly involved in the growth, may become solidified or destroyed from (*a*) collection of secretion within the bronchial tubes which are shut off from the trachea, causing dilatation of the tubes, and collapse of adjacent lung, (*b*) broncho-pneumonic obstruction of lung ensuing upon collapse, (*c*) inflammatory destruction of lung, secondary to pressure upon the pulmonary nerves. (Budd).[1]

DIAGNOSIS.—The diagnosis of tumor of the mediastinum, as soon as the attention is seriously drawn to the case by symptoms, is not as a rule difficult. In the first place pressure-signs and symptoms are incompatible with any form of pleuritic disease or pulmonary consolidation, nor are they markedly present even in extreme pericardial effusion. The conditions which most simulate mediastinal tumors are abscess, and syphilitic stricture of a main bronchus. The temperature, hectic phenomena, possible history of injury, character of pain and attendant inflammatory phenomena, will usually clear up the diagnosis in cases of abscess.

The distinction of syphilitic stricture of the main bronchus or lower trachea, from an obscure mediastinal tumor of small dimensions strangulating these parts, is most difficult. Still the limitation of pressure signs to one system, and the absence of any discoverable physical signs of tumor, together with the history of syphilis, would at least sufficiently clear up the diagnosis to suggest a definite treatment, the effects of which might perhaps, but not necessarily, throw further light upon the case.

Having arrived at the conclusion that there is a tumor present in the mediastinum, we have yet to determine whether it be aneurism or growth; and if the latter, what kind of growth.

The following are the chief points in favor of a mediastinal tumor being growth rather than aneurism.

1. The age of the patient, if under twenty-five. 2. The presence or history of tumors in other situations. 3. The absence of marked disease of the vessels. 4. The absence of characteristic pulsation or bruit; es-

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[1] On some of the effects of primary cancerous tumors within the chest, Medico-Chirurgical transactions, vol. xiii., p. 215.

pecially when combined with, 5, The presence of decided venous obstruction; and 6, Extensive area of superficial dullness.

In endeavoring to come to a diagnosis as to the nature of the growth present, the following points may be usefully remembered:

1. If the disease be primary in the mediastinum, it will be most probably lymphoma. 2. The younger the patient the more likely is the growth to be of this nature. 3. The presence of enlarged movable glands in the neck or other parts of the body is in favor of lymphoma. 4. If the disease be secondary to a growth elsewhere, it will be of the same nature as the primary disease. 5. So far as invasion of the lung goes, this feature is common to all growths.

TREATMENT.—No treatment has yet been found to influence the progress of mediastinal growths. The more distressing symptoms may be relieved by antispasmodic and sedative remedies. An effusion into the pleura should not be interfered with, unless it be distinctly increasing dyspnœa, its tendency being to retard the progress of the growth.

CHAPTER XXXV.

HYDATID OF THE LUNG.

NEXT to the liver the lung is the most frequent seat of hydatid tumor. The disease, however, is only rarely met with in this country, but in Australia it is much more prevalent.[1]

The disease is most prevalent in the male sex. It may occur at any age, and is nearly twice as frequent in the right as in the left lung: this preponderance in favor of the right side being most marked amongst cases of basic distribution. Sometimes the acephalocysts are multiple and affect both lungs. The bases or central portions of the lungs are most commonly affected, but one or both apices may be attacked. Sometimes the cysts have been found in the pulmonary arteries and right side of the heart, having been conveyed there from some more distant point. In the bronchial tubes the cysts, entire or broken, may be found on their way to expulsion.

The parasite finds its way to the lungs from the digestive canal, and through the pulmonary artery. The morbid anatomy of an hydatid tumor of the lung is after the same pattern as that of similar cysts elsewhere, the capsule being furnished by the more or less smoothed, thickened and condensed pulmonary tissue. The capsule of the pulmonary hydatid is thus in the lung, very vascular, and with it one or more bronchial tubes communicate.

SYMPTOMS.—The symptoms and signs of hydatid of the lung may be conveniently divided into:—1. Those presented by the tumor before rupture. 2. Those which occur during and after rupture.

Before rupture.—Unbroken cysts which have not attained considerable size, may give rise to absolutely no recognizable signs, and so slow and insidious is their growth, that even large tumors may exist for a long time unsuspected. Cough, hæmoptysis, pain, and dyspnœa, are the chief symptoms that may be present in this stage.

The cough is dry, teasing in character, sometimes attended with slight bronchial expectoration; occasionally it is distinctly paroxysmal, and has a laryngeal croupy character. Hæmoptysis is but slight, from a mere streak or staining of the sputum to a tea-spoonful or so, and is due to some active pulmonary congestion, set up by the growing parasite in its immediate neighborhood.

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[1] I am greatly indebted in my remarks on Hydatid of the Lung to Dr. Davies Thomas's (of Adelaide) admirable brochure, published by Mr. H. K. Lewis, 1884, and containing numerous tabulated cases and references.

Pain is not experienced except when the cyst has approached near enough to the surface to involve the pleura; but before this period an ill-defined uneasiness may be felt. The dyspnœa is inappreciable except in the cases of a large cyst, or one placed near the root of the lung. The centric pressure symptoms of an acephalocyst are, however, never very marked.

The physical signs before rupture may, in cases of small or deeply seated cysts, be completely obscured. Over tumors of larger size and nearer the surface, a certain degree of fullness may be observed with effacement of the intercostal spaces over a limited area. Percussion dullness, having a very definite and circular outline, is obtained over this area, beyond which there is normal or modified pulmonary resonance. A certain degree of elasticity may be appreciated on percussion over the most central point, amounting possibly to hydatid thrill.[1] Over the region of dullness, vocal fremitus and respiratory murmurs are enfeebled or absent. There is more or less displacement of the heart and other organs. In some cases pleuritic friction may be present over the tumor.

In association with these more or less positive signs and symptoms, there are the important facts of the absence of pyrexia, the but slight interference with general health and nutrition, and the very insidious and ill-defined onset of the disease. The position of the physical signs is sometimes of value in diagnosis, when they present in the mammary or axillary, infra-scapula, or some other central or marginal portion. The removal through a fine trochar of the characteristic hydatid fluid of low specific gravity, 1005-7, rich in chlorides and free from albumen, containing possibly some hooklets, will completely solve the diagnosis. This step, however, should not be taken until the diagnosis is so far arrived at as to prepare the practitioner for the rapid removal of a considerable quantity of fluid: and this for reasons to be presently mentioned.

Rupture of the cyst.—Spontaneous rupture of the cyst into the bronchial tubes takes place in about half the cases, into the pleura in five per cent., and more rarely into the pericardium.

Sudden pain, intense dyspnœa, and the expectoration of a large amount of watery, blood-stained fluid are the symptoms which immediately attend upon rupture of the sac into the lung. In some cases the lungs are so completely flooded as to overwhelm the patient and cause immediate death. In other cases the churning and rattling of the fluid and air in the chest can be heard, and after a desperate struggle the bronchial tubes become sufficiently cleared, when rapid amendment of symptoms takes place. Together with the fluid a greater or smaller number of daughter cysts may be expelled, and sometimes immediately, but more often at a subsequent date, the cyst wall may be expelled, entire or in fragments, with symptoms of threatened suffocation.

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[1] "Hydatid thrill" is, however, a very misleading term. I have felt the sign most perfectly elicited in a case of hydro-nephrosis, and also well-marked at the superior level of the fluid in pyo-pneumothorax.

It frequently happens that with these alarming symptoms the real nature of the disease is for the first time recognized, the expectoration of the hydatid membrane or daughter cysts being especially characteristic.

Hæmoptysis, much more profuse in quantity than in the earlier stages of the disease, may occur at the time of rupture and at any subsequent period, the hemorrhage being due to the tearing of the cyst membrane at the moment of collapse away from its vascular capsule, from which at the same time the support and even pressure[1] of the distended cyst is suddenly removed. Hæmoptysis is thus one of the most constant and important symptoms of hydatid of the lung, occurring in about four-fifths of the cases at some period: as a rule only slight in degree at the earlier stages, but often very severe and repeated after the rupture of the sac.

Of the large proportion of cases in which death is not caused by suffocation at the time of rupture, many (some sixty per cent.) recover after a time, during which they present more or less distinctly the physical signs and symptoms of pulmonary abscess. Of these cases also in which spontaneous rupture of the cyst does not take place, in a certain number, death of the parasite takes place with shrivelling of the sac and partial absorption or inspissation of its fluid contents. Many of these cases are preceded or followed by suppuration of the capsule, and thus pulmonary abscess is formed, and sooner or later discharged with shreds and débris of the cyst. When the cyst is small it may simply shrivel and become infiltrated with salts, forming a contracted obsolete cyst in the lung, and giving rise to no symptoms.

In those cases in which death of the parasite and partial separation from the capsule occur, a very fœtid abscess may result from decomposition of the sac.

The emaciation, hectic, occasional hæmoptysis, severe cough, and often profuse expectoration that characterize the later stages of hydatid disease, in combination with the physical signs of excavation of the lung and of great bronchial irritation, especially on the side affected by the parasite, are at first sight very suggestive of pulmonary phthisis. A careful examination into the history of the case, however, including the sudden commencement of expectoration as a copious outburst of blood-stained, watery or semi-purulent fluid, and the discovery of the physical signs of cavity in some situation unusual in phthisis, will arouse suspicions as to the true nature of the case, and a careful daily observation of the sputum may lead to the discovery of membranous shreds or the more typical "gooseberry skins," the structure of which microscopic examination will reveal to be that of hydatid cysts.

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[1] Dr. Thomas has ascertained the intra-cystic pressure in one hydatid of medium size to be from ten to twelve inches of water, but in another case in which the cyst was large enough to bulge, and therefore be compressed by the thoracic wall, the intra-cystic tension amounted to twenty-five inches during inspiration, thirty inches during expiration, *op. cit.*, p. 21.

TREATMENT.—Medicinal remedies in the early stages of hydatid of the lung are completely useless. In the later stages when the condition is practically that of abscess of the lung, bark and mineral acids, with a generous supporting diet are required.

In cases of large unruptured cysts, if left untreated, there is great danger of the patient being suffocated by the sudden rupture of the cyst flooding the bronchial tubes, or of a virulent pleurisy being set up by rupture of the sac into the pleural cavity.

On the other hand operative treatment must in this stage be very cautiously undertaken. The operative measures for this stage are of two kinds:—1. Puncture with a fine or medium-sized trochar connected with an aspirator or syphon tubing, *i.e.*, paracentesis. 2. The more radical operation of incision and evacuation of the entire parasite, *i.e.*, thoracentesis. The danger connected with the apparently simple operation of paracentesis arises from the elasticity of the sac, and its commonly containing daughter-cysts; the elasticity of the sac giving it a tendency to retract and elude the trochar, the watery fluid thus escaping into the capsule and obtaining exit through the bronchial tubes as in spontaneous rupture. The trochar may also become occluded by secondary cysts and thus fail to empty the sac, which again discharges its contents into the capsule.

A bold introduction of the trochar deeply into the cyst and the provision of a second side opening to the canula will tend to avert both these misfortunes.

The radical operation of incision and removal of the cyst and its contents though the external opening enlarged by dressing forceps, commends itself as the most rational treatment, and in a case in which pain and auscultatory evidences of pleurisy had been observed, leading to the inference that adhesions were present, it would probably be the safest treatment, but not otherwise. In a large proportion of cases, however, the cysts are two deep for any operative treatment, and in a good proportion of such cases spontaneous rupture leads to the expulsion or obsolescence of the sac.

After rupture each case must be treated on its merits, too hasty an operative interference being, however, much to be deprecated. In cases where the patient is notably losing ground from hectic and profuse or fœtid expectoration, the question of operation must be carefully and in good time considered. The operative measures to be adopted would be identical with those for abscess of the lung.

APPENDIX.

INTERNATIONAL NOMENCLATURE OF PHYSICAL SIGNS.

APPENDIX :—NOTE ON INTERNATIONAL NOMENCLATURE OF PHYSICAL SIGNS.

The following list of auscultatory sounds was presented by Dr. Austin Flint, Chairman of the Committee appointed at the International Congress in London, in 1881, to report to the Medicine Section of the next Congress on a uniform nomenclature of auscultatory sounds in the diagnosis of diseases of the chest.

The Committee nominated were Dr. Austin Flint of New York, Professor Ewald of Berlin, Professor D'Espine of Geneva and Drs. Mahomed and Douglas Powell of London. The subjoined lists were drawn up after much consideration and references to many physicians and teachers of medicine in this and other countries. Although close unanimity existed amongst the members of the Committee, the lists were only presented to the Congress at Copenhagen in 1884 as provisional, with the view of eliciting further suggestions and discussions.

I. PALPATION.

ENGLISH & AMERICAN LISTS.	GERMAN LIST.	FRENCH LIST.
1. Vocal fremitus.	Stimmfremitus oder Vocalfremitus.	Vibrations thoraciques.
2. Rhonchal fremitus.	Rhoncofremitus oder Bronchialfremitus.	(Not included.)
3. Friction fremitus.	Pleuritisches resp. Pericarditisches Reiben.	Frottement.

II. PERCUSSION.

ENGLISH & AMERICAN LISTS.	GERMAN LIST.	FRENCH LIST.
1. Tympanitic resonance.	Tympanitischer Schall.	Son tympanique : (*a*) grave ; (*b*) élevé.
2. Amphoric resonance.	Metallklang.	Bruit de pot fêlé.
3. Diminished resonance ; dullness.	Dämpfnung.	Submatité.
4. Absence of resonance ; flatness.	Absolute oder starke ; Dämpfnung.	Matité
5. Increased resonance.*	Hypersonover Schall.	Son pulmonaire exagéré.
6. Bell sound.	(Not included.)	(Not included.)

III. AUSCULTATION.

A. *First Group. Varieties of Breath Sounds.*

ENGLISH & AMERICAN LISTS.	GERMAN LIST.	FRENCH LIST.
1. Exaggerated. Syn. : Puerile ; compensatory ; supplementary.	Verschärftes Athemgeräusch.	Respiration exagérée.
2. Diminished. Syn : Feeble ; weakened vesicular murmur.	Abgeschwächtes Athemgeräusch.	Respiration diminuée.
3. Suppressed. Syn. : Absence of breath sound.	Fehlendes Athemgeräusch.	Respiration supprimée.
4. Prolonged expiration, general or local.†	Verlängertes Expirium.	Expiration prolongée.

5. Interrupted inspirated. Syn.: Jerking, wavy, cog-wheeled.	Saccadirtes Athemgeräusch.	Respiration saccadée.
6. Tubular. Syn.: Bronchial; high-pitched blowing.	Bronchiales Athemgeräusch: (*a*) stark; (*b*) tief.	Souffle bronchique: (*a*) superficiel; (*b*) profond; (*c*) doux; (*d*) fort.
7. Vesiculo-tubular. Syn.: Broncho-vesicular; harsh, coarse, sub-tubular.‡	Unbestimmtes Athemgeräusch.	Respiration rude; broncho-vesiculaire.
8. Amphoric.	Amphorisches Athemgeräusch.	Souffle cavitaire amphorique, avec écho, musical ou métallique.
9. Cavernous.	(Not included.)	Souffle cavitaire simple, sans écho, musical.

B. *Second Group. Adventitious Sounds.*

1. Rhonchi. Dry musical sounds: (*a*) sonorous; (*b*) sibilant.	Rhonci. Trockene Rasselgeräusche: (*a*) sonor oder schürrens; (*b*) sibilant; oder pfeiffend.	Rhoncus. Râles secs: (*a*) ronflants; (*b*) sibilants.
2. Stridor.	Stridor. Stenosengeräusch.	Sifflement laryngé. Bruit de cornage: (*a*) trachéal; (*b*) bronchique.
3. Râles. Syn.: Bubbling râles—(*a*) Medium; (*b*) large; high or low in pitch.§	Feüchte Rasselgeräusche: (*a*) grossblasige; (*b*) mittelgrossblasige; (*c*) kleinblasige.	Râles humides: (*a*) gros; (*b*) moyens; (*c*) fins; (*a*) avec timbre bronchique; (*b*) sans timbre ou simples.
4. Gurgling.	Höhlenrasseln.	Gargouillement.
5. Clicking.	(Not included.)	Craquements: (*a*) secs; (*b*) humides.
6. Crepitation. Syn.: Crepitant râles.	Knisternde Rasselgeräusche; Knisterrasseln.	Râles crépitants.
7. Metallic tinkling.	Metallische Rasselgeräusche; Metallklang.	Tintement métallique.
8. Splash.	Succussionsgeräusch.	Succussion hippocratique.
9. Friction: (*a*) dry; (*b*) moist.	Reibegeräusch.	Frottement.

C. *Third Group. Varieties of Voice Sounds.*

1. Increase of vocal resonance.	(Not included.	Voix exagérée.
2. Diminution or absence of vocal resonance.	Abschwächung und Fehlen der Stimme.	Voix diminuée.
3. Bronchophony.	Bronchophonie.	Bronchophonie.
4. Pectoriloquy.	Pectoriloquie.	Pectoriloquie.
5. Ægophony.	Ægophonie.	Ægophonie.

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* American: Increased or vesiculo-tympanitic resonance.

† American: Prolonged expiration; high or low in pitch.

‡ American: Broncho-vesicular. Syn.: Vesiculo-tubular.

§ Moist or bubbling râles—(*a*) large, (*b*) medium, (*c*) small, ; high or low in pitch.

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